Dissertation on

"A PROSPECTIVE STUDY ON RETINAL DEGENERATIONS AND COMPLICATIONS IN 100 CASES OF PROGRESSIVE PATHOLOGICAL MYOPIA AND ITS MANAGEMENT"

Submitted in partial fulfilment of requirements of

M.S. OPHTHALMOLOGY

BRANCH - III

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MADRAS MEDICAL COLLEGE

CHENNAI- 600 003

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

MAY 2018

CERTIFICATE

This is to certify that this dissertation entitled **"A PROSPECTIVE STUDY ON RETINAL DEGENERATIONS AND COMPLICATIONS IN 100 CASES OF PROGRESSIVE PATHOLOGICAL MYOPIA AND ITS MANAGEMENT"**is a bonafide record of the research work done by Dr. J. Aarthy Grace, post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2015-2018.

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
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Dear Dr.Aarthy Grace.J,

The Institutional Ethics Committee has considered your request and approved your study titled "**A PROSPECTIVE STUDY ON RETINAL DEGENERATIONS AND COMPLICATIONS OF 100 CASES OF PROGRESSIVE PATHOLOGICAL MYOPIA AND ITS MANAGEMENT" - NO.03022017 (II)**

The following members of Ethics Committee were present in the meeting hold on **21.02.2017** conducted at Madras Medical College, Chennai 3

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We approve the proposal to be conducted in its presented form.

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Place : Chennai Dr. J. AARTHY GRACE

Date :

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PART I

INTRODUCTION

INTRODUCTION

The most important sense organ of the human body is Eye. The responsibility of visual function is primarily carried out by Retina. Pathological changes of the retina is one of the cause of irreversible blindness. Myopia is one of the cause of impaired visual acuity among school going children as well as those in adults. Most commonly retinal degeneration and retinal detachment is seen with the myopic patients.

Among the special senses and the central nervous system, retina is a unique and a complex element. It can be readily viewed during life, and it is a transparent structure so that alterations within and adjacent structures can be observed in vivo.

Retina has been an area of ever - increasing importance in science as a whole and in the field of ophthalmology due to its unique structure, organisation and function.

Methods to view the retina have steadily improved during the century since the principle of ophthalmoscopy was presented by Von-Helmholtz. Thereby the techniques of ophthalmoscopy and bio-microscopy facilitate the clinical examination of the entire retina in detail.

The accurate and detailed knowledge of the topography of the retina, the anatomical relationships, and the developmental variations of various degenerations affecting the retina help in the proper interpretation of findings.

Pathologic myopia is an eccentric group wherein the myopia is likely due to a disease rather than a biologic variation. The myopic eyes show excessive axial length with increased scleral expansion, dehiscence and posterior staphyloma formation. The global expansion of the eye is a slow process that occurs during a person's life resulting in blinding complication. The myopia of – 6.D to an excess of -40.00 D comes under the criteria of pathologic myopia. The recent technologies like B-scan, ICG, FFA and OCT help us to understand and monitor the underlying pathology and the structural alterations better.

HISTORICAL REVIEW

HISTORICAL REVIEW

The word **Myopia**is derived from the Greek word **Muopia** which means contracting or closing the eyes.

ARISTOTLE – first person who noted the tendency of myopes to blink and write in small script

FRANS CORNELIS DONDERS (1818-1889) – first person to analyse the various types of refractive error.

NEWTON (1704) – Noted that axial length is the sole determinant of refraction.

PLEMPIUS (1632) – Proved that myopes the axial length is more.

SCARDIA (1801) – The first person who anatomically described posterior staphyloma.

VON AMMON (1832) – Pointed out that posterior staphyloma was due to distension of the posterior pole.

VON GRAEYE and **VON JAEGER** (1854) - Postulated the association of myopia and posterior staphyloma.

ARLT (1856) – association of myopia with axial elongation.

REHSTEINER (1928) – Noted the peripheral degenerative changes in pathological myopia.

STENSTROM (1946) – Measured the ocular axial length directly by X-Rays.

PERCIVAC (1987) – axial length in addition to pathology is a factor associated with retinal detachment.

KREMER and Co-workers – high myopes showed the presence of multiple atrophic retinal holes in the posterior pole.

BURTON – refractive error and lattice degeneration on detachment.

MORITA and Coworkers – risk factors associated with retinal detachment.

ANATOMY AND TOPOGRAPHY OF RETINA

CENTRAL RETINA

The retina is a delicate and thin layer of nervous tissue that has the surface area of about 266 mmsq. The major parts of the retina are the optic disc, the area centralis with fovea & foveola, the retinal blood vessels, the peripheral retina and the ora serrata. The retina is thickest near the optic disc, measuring 0.56 mm, becomes thinner towards the periphery.

THE OPTIC DISC

The optic nerve head, the collection point for the axons of its ganglion cells is the optic disc. The disc is circular to slightly oval structure (1.5mm), which contains a depression in the centre, the physiological cup. The centre of the optic disc is about 4mm nasal to the fovea. The optic nerve head receives about 1.2 million retinal axons, which turn at about right angle to make an entry into the optic nerve. Its centre is 3.42 mm medial and 0.1 mm inferior to fovea. Its vertical diameter is 1.86 mm and horizontal diameter is 1.75 mm (**straatsma,Foos,Spencer**, 1969) and it lies 27 mm from the nasal and 31 mm from the temporal limbus, The axons pass through the multi lamellar fenestrations of the collagenous lamina cribrosa which occupies the posterior scleral foramen. Optic disc head is supplied by branches of short ciliary arteries, except the of nerve fiber layer which is supplied by the central retinal artery.

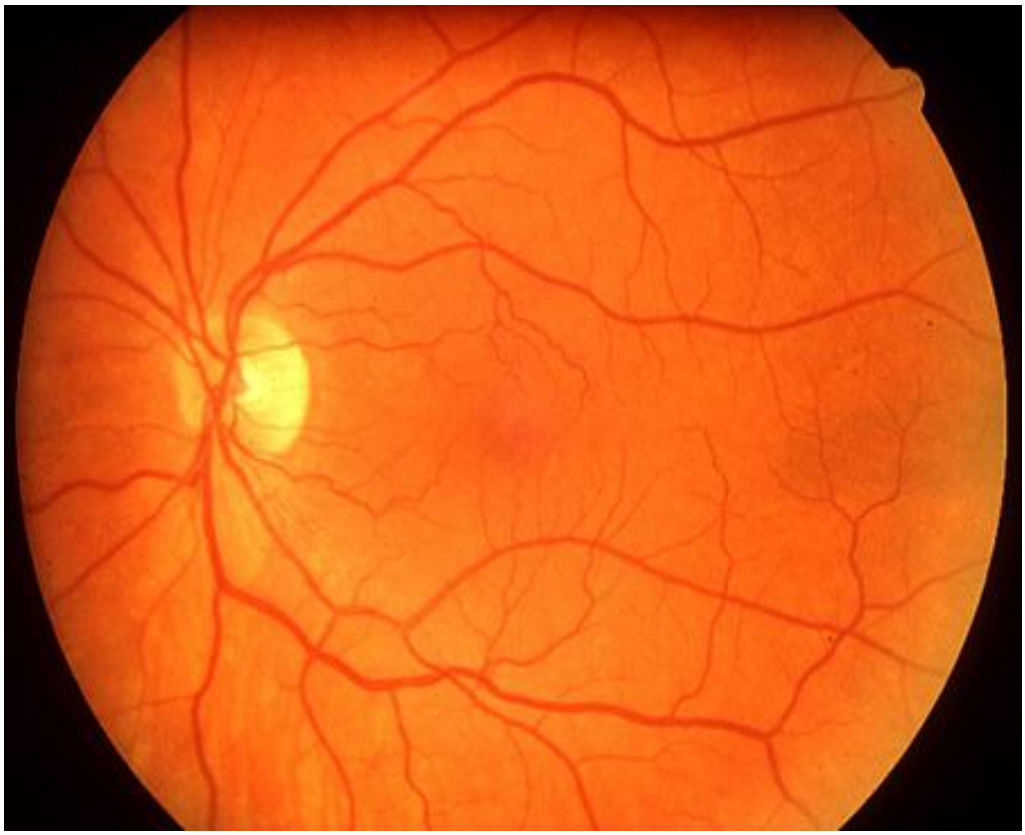
THE AREA CENTRALIS

The central retina is divided into the fovea and foveola, with a para foveal and perifoveal ring around the fovea. This region of the retina located in the posterior fundus temporal to the optic disc and is demarcated by the upper and the lower arcuate and temporal retinalvessels approximately and has an elliptical shape. With an average diameter of about 5.5 mm the area centralis corresponds to approximately 15 degrees of the visual field.

FOVEA

Located at the posterior pole of the globe, 4mm temporal to the centre of the optic disc and about 0.8 mm below the horizontal meridian. Diameter 1.85mm, thickness 0.25mm. The downward sloping border meets the floor of the foveal pit and is known as clivus.

NORMAL FUNDUS



FOVEOLA

It is 0.35mm in diameter and 0.13mm in thickness. Represents the area of highest visual acuity.

MACULALUTEA

Is the oval zone of yellow colouration within the central retina. Yellow colouration probably is due to the presence of carotenoid pigment, xanthophyll in the ganglion and the bipolar cells. **(Tripathi & Tripathi 1984).** Pigment epithelium in the posterior fundus is less granular than at the periphery. Concentration of cone is maximum in the central retina. Ganglion cell layer is seen in two layers at the temporal side of the optic disc and is about 6-8 layers at the edge of the foveola. The ganglion cell layer is absent at the foveola of the optic nerve head.

PERIPHERAL RETINA

The peripheral fundus is defined as the area anterior to the scleral entrance of the vortex to the middle of pars plana. There are four regions in peripheral retina – near periphery, mid periphery, far periphery and the ora serrata.

NEAR PERIPHERY – Is a circumscribed lesion around the area centralis measuring 1.5 mm

MID PERIPHERY – Located 3 mm around the zone of near periphery.

FAR PERIPHERY – Extends 9-10 mm on the temporal retina and 16 mm on the horizontal meridian towards the nasal side.

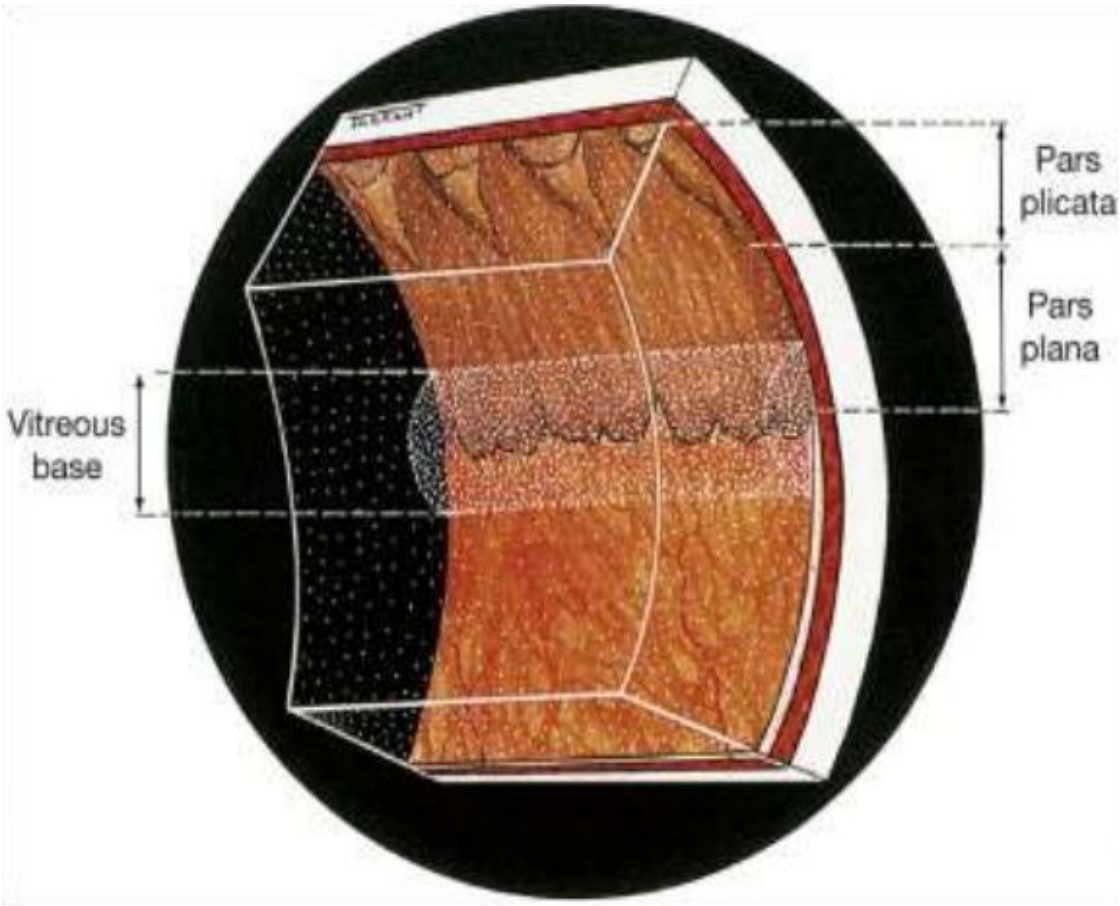
ORA SERRATA :

The peripheral most edge of the retina is the ora serrata. It is the junction between the pars plana retina which is multi layered anad the non- pigmented epithelium of the ciliary body which is monolayered.

VITREOUS BASE

Vitreous base is approximately 3.2 mm wide slightly wider nasally and narrow temporally. Anterior portion of the vitreous base is located in the zone between the ora serrata and the origin of the anterior hyaloid membrane. Anterior vitreous base is generally conformed to the contour of the ora serrata.The area of the vitreoretina near the posterior portion of the vitreous base is the strongest portion that extends posterior to the ora serrata.

VITREOUS BASE



EPIDEMIOLOGY

PREVALENCE OF MYOPIA

Prevalence of myopia varies with age, sex and other factors. Most infants reach emmetropia by 2-3 years of age. School age and young adults has the highest prevalence of myopia reaching 20%-25% in mid to late teenage population and 25%-35% in young adults. Studies show a slightly higher prevalence of myopia in females than in males. The prevalence of myopia increases with income level and educational attainment and it is highest among the persons who work in occupation that requires great deal of near work.

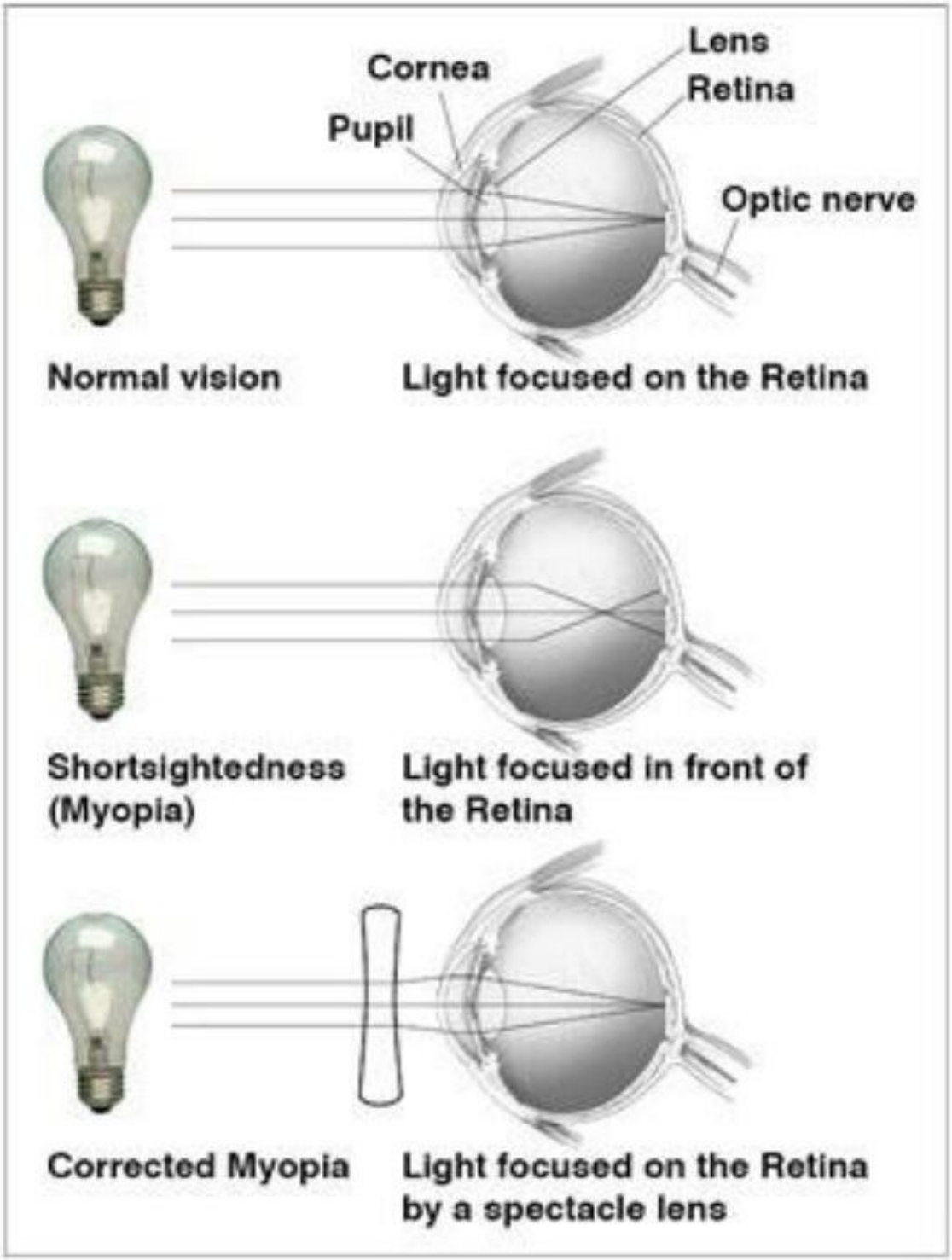
Among children in India, **Shulka** found myopia to increase from below 5% at 5 years to 20% at 20 years. **Mc laren** compared 2 groups of Indian children in which one group who had nutritional supplement showed better general development , also had slightly higher prevalence of myopia

OPTICS OF THE EYE

Emmetropia : The condition in which the parallel beam of light come to focus on the retina, with the eye at rest. At birth the average axial length is 18 mm, and infant eye undergoes rapid growth in the first few years of life to reach an axial length of 23 mm.

Ammetropia: The condition in which the incident parallel rays of light do not focus upon the light sensitive layer of retina.

OPTICS OF THE EYE



TYPES OF AMETROPIA

Axial ametropia:

Abnormal increase in the length of an eyeball (an 1mm elongation produces approx 3D of myopia)

Curvature ametropia :

Abnormal curvature of the refracting surfaces of the cornea or lens (1 mm change in the radius of curvature of the cornea produces a 6.00 D refractive error)

Index ametropia :

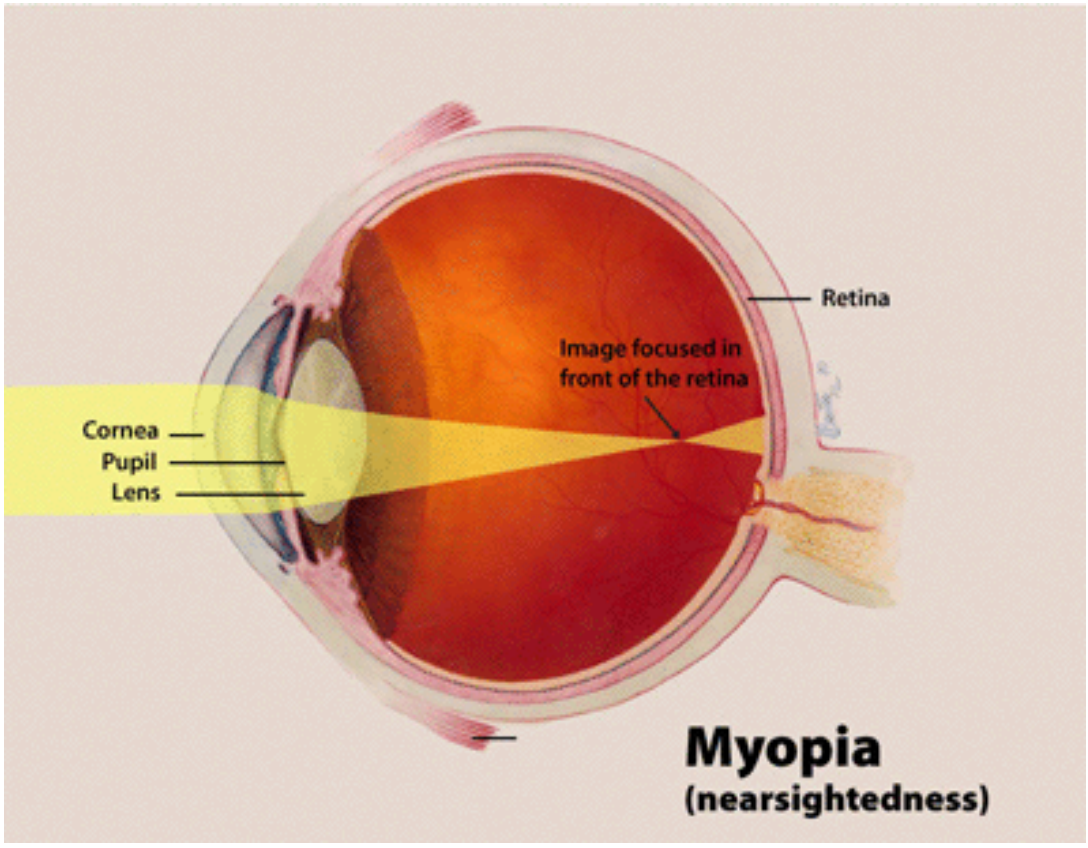
Abnormal refractive indices of the media.

MYOPIA

DEFINITION :

Myopia is that form of refractive error wherein parallel rays of light come to focus in front of the of the retina when the eye is at rest.

MYOPIA


Retina
Image focused in front of the retina
Cornea
Pupil
Lens
Myopia
(nearsightedness)

CLASSIFICATION OF MYOPIA

Etiological Classification:

Axial Myopia:

It is the most commonest type seen. It is due to increase in the anteroposterior diameter of the eye. For every 1 mm increase will cause 3.00 D increase in myopia.

Curvature Myopia :

This is seen due to an increase in the curvature of the cornea or the surface of the lens.

Index Myopia:

Occurs due to the change in the refractive index of the media. Example – Myopia seen associated with cataract and diabetes.

CLINICAL CLASSIFICATION:

Simple myopia :

It is the physiological variant of the normal. This is a condition of limited progression. Simple myopia are of two types.

Physiologic Myopia:

Here each component of refraction lies upon its normal distribution curve. Postnatal development is normal. There is correlation failure between the total refractive power and a normal axial diameter. The heredity is multifactorial. Myopia of -3.0 Diopters and less is physiologic.

Intermediate Myopia:

There is increased expansion of posterior segment of globe. The entire posterior segment is involved. Generalised thinning of retinal pigment epithelium seen. Myopia upto -8.0 dioptres associated with associated with various fundus changes can be considered intermediate **(B.J.Curtin).**

Pathological Myopia:

It is also called as malignant myopia. Determined by hereditary and postnatal factors. There is excessive axial elongation of the eye and a number of ocular complications. Myopia of -6.0 Dioptres or more is considered pathological.

PATHOGENESIS

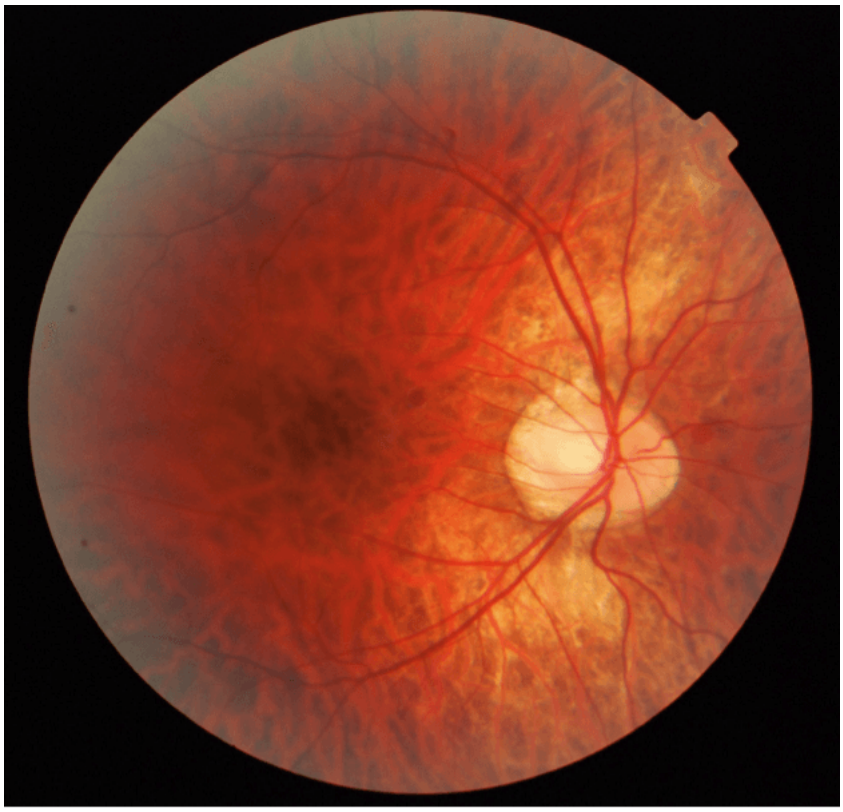
Pathologic myopia is characterized by degenerative changes seen occurring particularly in the posterior segment of a highly myopic eye. Often associated with lengthening of the anteroposterior axis of the globe. It connotes an extreme axial elongation in which the degenerative as well as vascular alterations are superimposed with each other.

The most common form of pathologic myopia is the isolated developmental form, Where as in simple myopia the myopic tendency decreased after puberty. In developmental pathologic myopia, the near sightedness may increase more rapidly during adolescence and the axial enlargement may slowly increase during adulthood to the 40s and 50s, with the eventual genesis of degenerative intraocular changes that leads to visual loss and possibly blindness.

Congenital axial pathological myopia may also occur and is frequently associated with other congenital defects such as colobomas and anomalies of pigmentation of the retina or choroid. It is closely associated with fundus conditions that resemble partial albinism.

Varying degrees of myopia commonly are associated with ROP, micropthalmia, microcornea, microphakia, bupthalmos, the tapetoretinal dystrophies and down syndrome.

TESSELATED FUNDUS



INHERITENCE

The pathogenesis of pathological myopia remains unclear. Previous reports have identified a locus for autosomal dominant pathologic myopia gene **18p11.31.** More recent findings posit the genetic heterogeneity of myopia by establishing linkage to a second locus at the **12q2123** regions. High myopia is more likely to develop in women than men, whereas the lower degrees of myopia are generally transmitted as a dominant trait. In higher degrees of myopia, which begin at a relatively early age, recessive transmission is more common. Anisometropia, an unequal degree of myopia in each eye, is the rule in most of high pathologic myopia, but gross inequalities greater than 3D are mostly unusual.

OCULAR CHANGES IN PATHOLOGICAL MYOPIA:

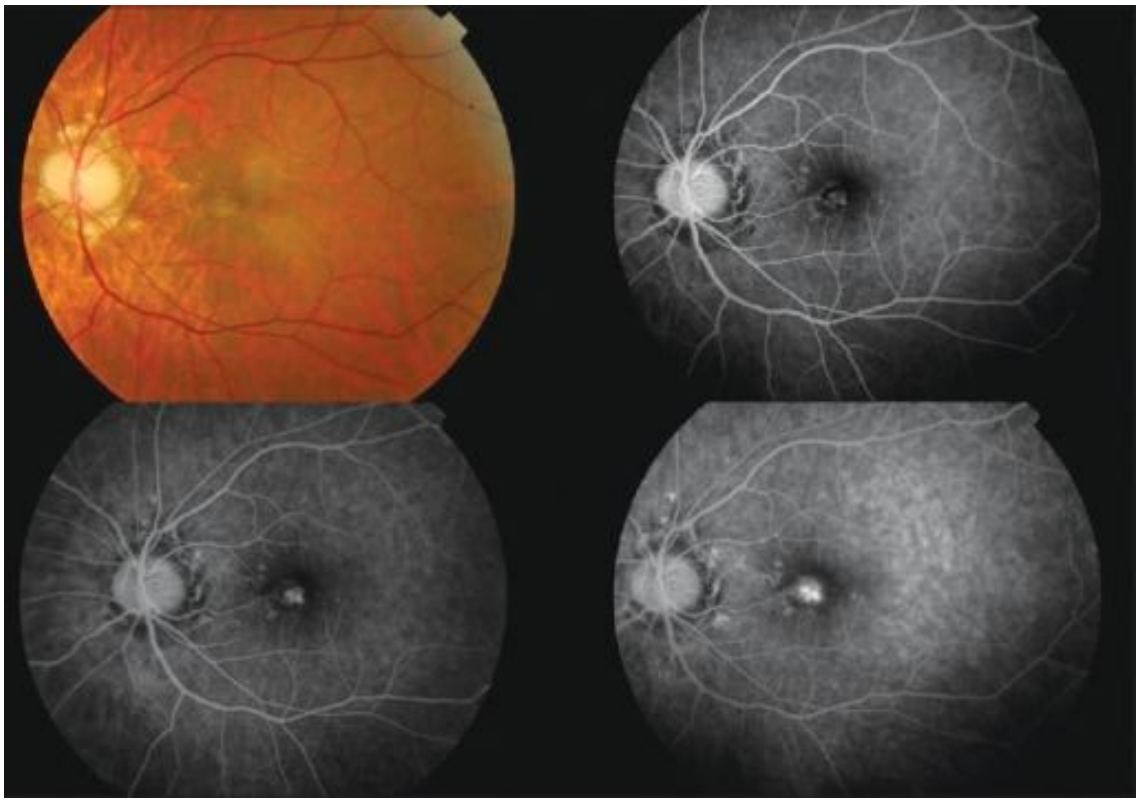
Clinically , a severe myopic eye generally appears large and prominent. The gross appearance of the highly myopic eye is egg or pear shaped and is significantly enlarged. The cornea may be abnormally flat, the anterior chamber is somewhat deeper than normal and the ciliary muscles are atropic. The ciliary muscle in a person with high myopia often is smaller than normal, probably because the myopic individual use the muscles of accommodation lesser.

CHANGES IN POSTERIOR SEGMENT:

The major changes are confined almost entirely to the posterior pole. The first to correlate the histologic changes in myopia with the ophthalmoscopic changes was by von grafe. These changes are summarized as follows:

i) **Scleral changes** - posterior enlargement of the globe and thinning of the sclera at the posterior pole with scleral ectasia and posterior staphyloma.

ii) **Changes in the epipapillary and peripapillary region** – oblique entrance of the optic nerve, tilted disc, myopic crescent, nasal super traction.



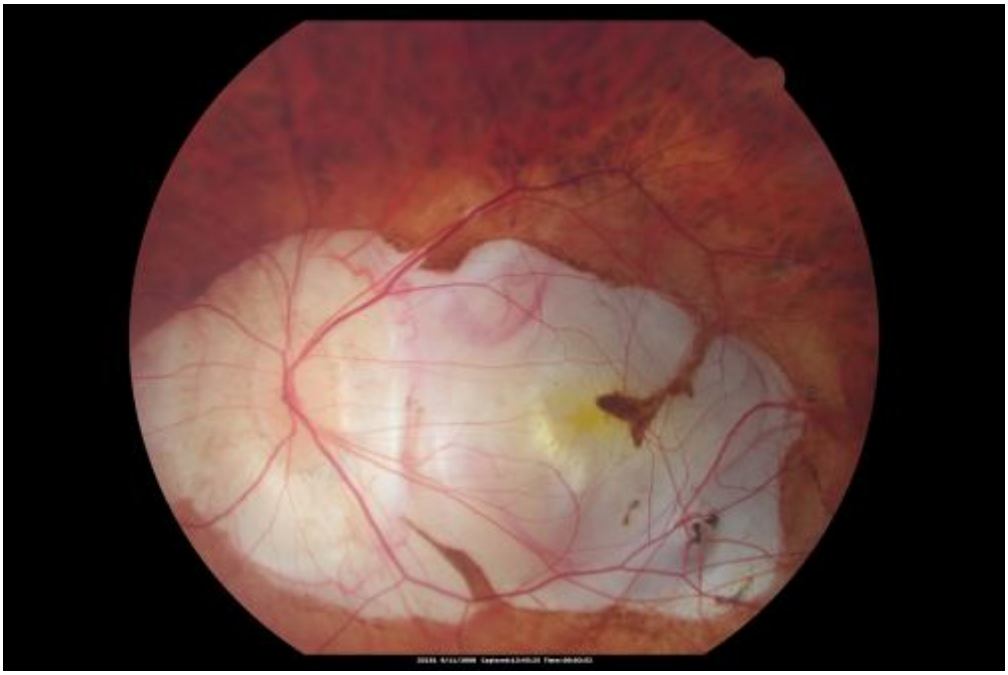
iii) **Changes in the choroid and retina**- atrophy and thinning, particularly affecting the posterior pole and the periphery. These changes include atrophy and or proliferation of the pigment epithelium, foster fuchs spot at the macula, retinal microcystoid degeneration , and rarely peripheral retinal break formation and subsequent detachment.

iv) **Degenerative changes in the vitreous**.

1). SCLERAL CHANGES

Scleral thinning with occasional formation of the posterior bulging or staphyloma of the sclera is common. The staphyloma may surround the optic nerve head and extend temporally to involve the posterior pole and sometimes also the equator. The normal sclera progressively thickens from the equator backward, becoming thickest at the posterior pole. In a globe with severe myopia the sclera becomes progressively thinner posteriorly in the peripapillary region. When present, a staphyloma is lined by a thin atrophic choroid, and the margins of the staphyloma usually has an abrupt edge.

POSTERIOR STAPHYLOMA



TYPES OF POSTERIOR STAPHYLOMA:

Mainly five primary varieties are seen. Their features are as follows

TYPE 1 :

The tessellation and pallor will extend over a horizontal elliptical area. Site is nasal to disc margin commonest type seen.

TYPE 2:

Is called as macular staphyloma. Extends from the optic nerve to the temporal aspect of macula.

TYPE 3:

It is the least common type. Involves a well circumscribed area around the disc called as peripapillary staphyloma.

TYPE 4:

Usually the Nasal or inferonasal aspect of the optic nerve head is involved. There is associated inversion of the retinal vessels. Hence also called as inverse myopia.

TYPE 5:

It usually shallow and involves an elliptical zone below disc. Commonly considered as a form of choroidal coloboma.

2) CHANGES IN THE EPIPAPILLARY AND PERIPAPILLARY REGIONS

Ophthalmoscopically, the optic nerve head of an acquired myopia is ovoid with the long axis of it in the vertical direction. Myopic degeneration usually makes their initial appearance in the crescent margin. In very severe cases entire peripapillary area can be involved. In a myopic eye the disc appears tilted with the temporal side flattened and is surrounded by a concentric or crescent shaped areas of relative fundus depigmentation.

The myopic crescent invariably occurs in later years in those patients with myopia greater than 6 D. The sclera is visible because of an absent pigment epithelium and choroid, Both of which fail to extend to the temporal margin of the disc. The crescent of acquired myopia are located temporally in approximately 80% of cases . In 10% of cases , crescent extends to become annular, surrounds the entire disc, sometimes it spreads to include a large area of the fundus with envelopment of the macular area. In rare instances, the myopic crescent is present on the nasal side of the disc (inverse crescent).

3.CHANGES IN THE CHOROID AND RETINA

Atrophy of the choroids occurring predominantly near the posterior pole is almost a consistent feature of severe pathological myopia, Initially the retinal pigment epithelium becomes attenuated and then the choroidal vessels become visible. Splits may develop in bruchs membrane. These form clefts (lacquer

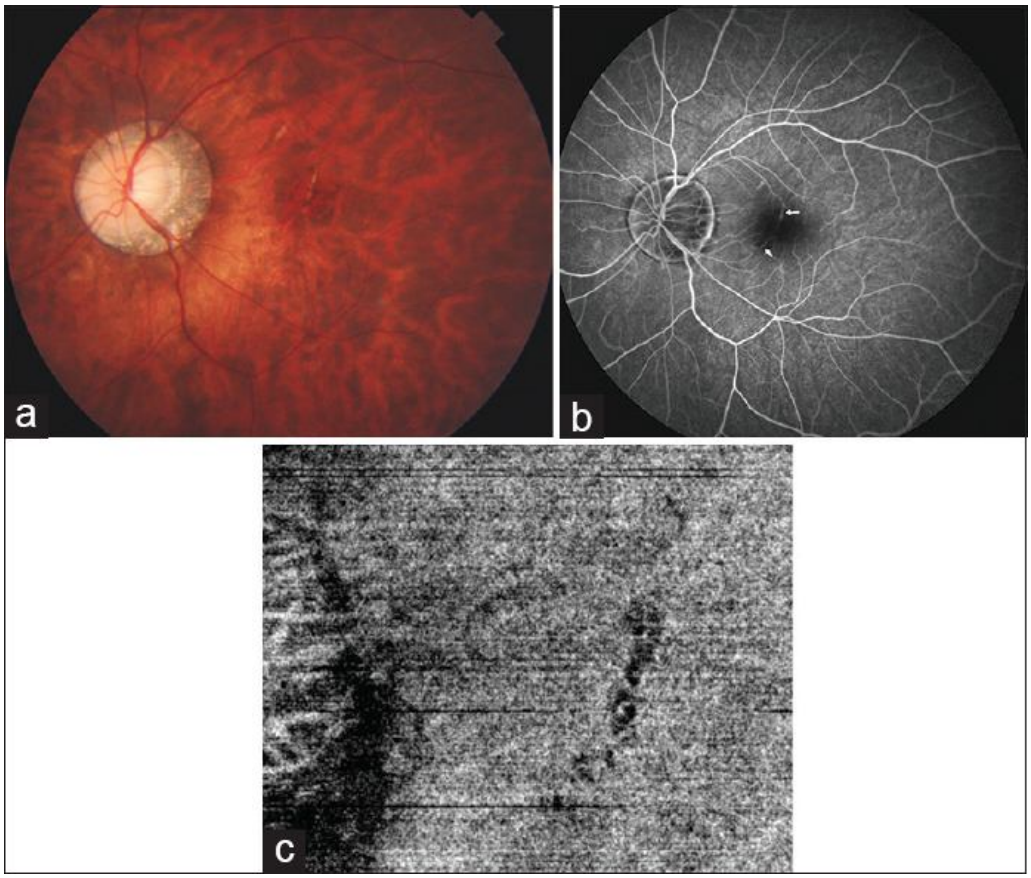
cracks or lightening figures), which has branches and have a reticular appearance. During the course of pathological myopia choroidal haemorrhages occur at the macula. They can be isolated or along with lacquer crack formation. The plane is between retinal pigment epithelium and lamina vitrea.

LACQUER CRACKS

The ruptures of the lamina vitrea is seen as lacquer cracks. This appears as yellow white lines across the posterior pole. Which is irregular in calibre. They are multipleand are horizontally oriented with a criss cross pattern.

These lesions are traversed by large choroidal vessels posteriorly. Inner layers of the retina is normal. Associated with concentric contraction of the field. Yellow blue colour vision deficiency is seen. If they are in macula, central vision is impaired. Along these lesions focal areas of chorioretinal atrophy are also seen.

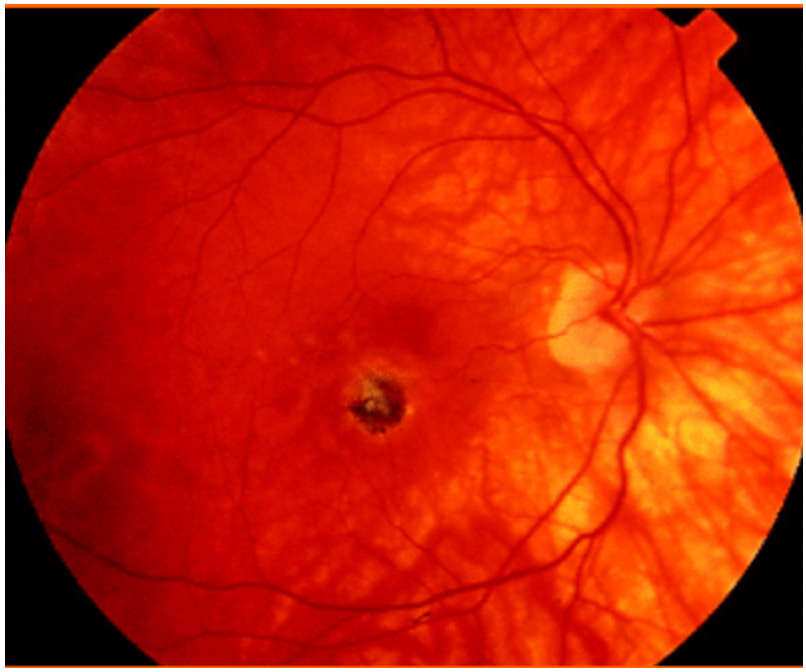
LACQUER CRACKS



FOSTER FUCH'S SPOTS:

Through the defect in lamina vitrea proliferation of choroidal fibrovascular tissues occurs and a firm adhesion develops between choroid and retina. This fibrovascular tissue can cause haemorrhage. There is a marked proliferation of overlying retinal pigment epithelium. Which forms unique well defined, elevated, black lesion at the posterior pole of eye foster fuchs spot.

FOSTER FUCH'S SPOTS:



CHOROIDAL NEOVASCULARISATION

IN MYOPIA

DEGENERATIVE CHANGES IN THE VITREOUS :

Changes in the vitreous includes liquefaction, microfibrillar degeneration and formation of opacities and floaters (muscae volitantes) . Posterior detachment of the vitreous commonly occurs, probably because of stretching of the enlarged globe, leaving a gap between the posterior vitreous and the posterior pole of the eye.

DEGENERATIONS OF THE PERIPHERAL RETINA

i) Retinal hole

Is a more advanced tropic lesion, manifest grossly as a round complete retinal break without detectable flap or operculum. These holes are commonly found in the anterior zone, mostly seen in an area of relatively normal retina.

ii) Cystoid degeneration:

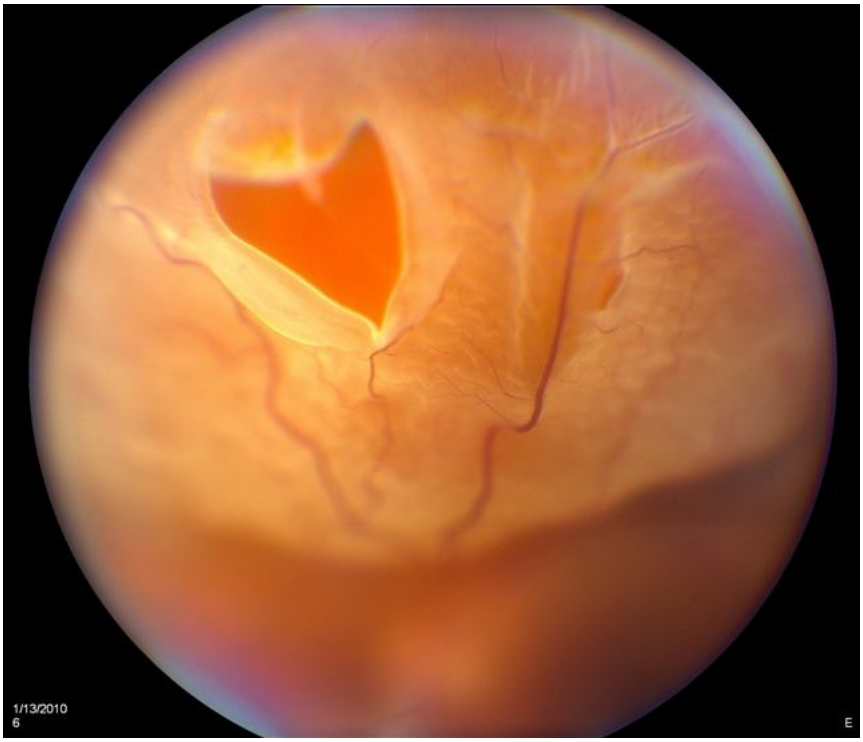
Inner wall of single cyst might be absent or broken giving the appearance of retinal hole. This is a pseudo-hole since the outer wall of the cyst is intact.

Another type of cystoid degeneration is the reticular cystoid degeneration of the peripheral retina , is almost invariably located posterior and continuous with the typical cystoid degeneration. Retinal cystoid degeneration is present in 18% of adult patients, commonly seen in the infero – temporal quadrant.

iii) Retinoschisis :

The splitting of the neural layers of the retina generally occurs in the outer plexiform layer in this condition. Typical retinoschisis is a more extensive tropic process whichpresents as a round or oval area of retinal splitting with a smooth fusiform elevation of the inner layer and its blood vessels.

RETINAL TEAR



RETINOSCHISIS

iv) Paving stone degeneration :

It has one or more discrete rounded foci of depigmentation and retinal thinning which is located between the ora serrata and the equator. The lesion are yellow white and it reveals the underlying choroidal vessels and often has a

pigmented margin. The basic lesion is rounded foci which may merge to form larger lesions with scalloped margin and a incomplete septum. Histologically it is characterised by the loss of retinal pigment epithelium in outer retina with adhesion of the inner retina to the bruch's membrane.

Paving stone degeneration does not predispose to retinal break or retinal detachment.

v) Chorioretinal degeneration :

It is always seen extending to the fundus periphery. It is most severe in the retina adjacent to the ora serrata. It spreads posteriorly and merges into the normal healthy retina without definite demarcation. Chorioretinal degeneration is generally associated with cystoid degeneration, both conditions more or less occupies the same area. The ophthalmoscopic appearance of chorio-retinal degeneration is graded as mild, moderate or severe.

The changes are always severe closer to the ora serrata and mild posteriorly. Peripheral chorio-retinal degeneration begins to appear in the fourth decade of life and increase with the age. Males affected more than the females.

vi) Chorio-retinal atrophy:

Is seen as areas of retinal and choroidal thinning. Pigment proliferation, migration of pigment in the retina are present near and around the edges of the lesion where thecentre is pale and dirty grey. Atrophy of the inner choroidal layer cause exposure of the large choroidal vessels.

vii) Pigmentary degeneration :

Of the various types of peripheral changes, pigmentary degeneration is the least studied and less understood lesion. The pigmentation may vary from a fine diffuse darkening of the fundus to large discrete clumps. Pigment may be seen as scattered clumps, granules or as localized clumps or may be diffusely present. Pigmentary degeneration has tendency towards bilaterality and apparently there is no sex preference. Age does not seem to be an important factor. It has tendency to be found with white without pressure or lattice degeneration and silent retinal breaks.

viii) White without pressure:

They are seen as circumferentially arranged geographic white or grey areas . They may be flat or elevated. The most common site is inferior quadrant, posterior to the equator. The surface is covered by glistening yellow white dots and fine lines.

ix) White with pressure :

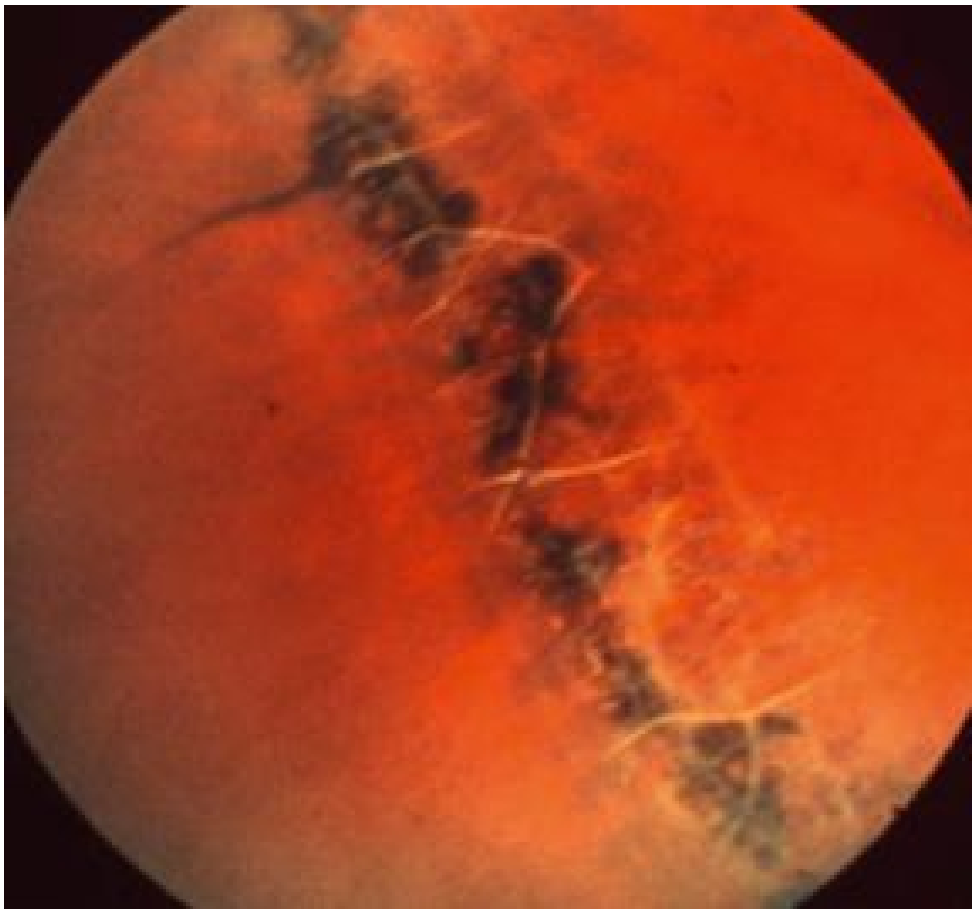
Usually found in area of lattice and small retinal breaks. Also seen in eyes with vitreous and retinal detachments. They are degenerative changes and are benign lesions.

x) Lattice Degeneration :

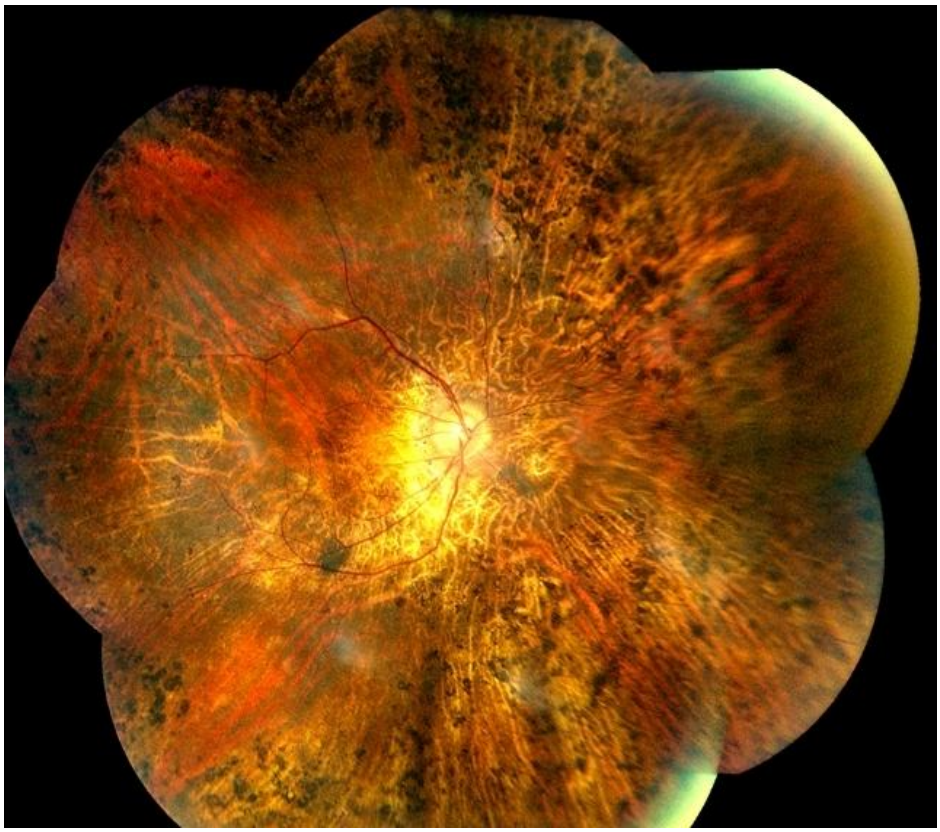
Most common lesion linear or spindle shaped and are seen at or peripheral to equator.They have a sharp demarcation and are circumferentially oriented. Variable amount of pigment proliferation is also seen. Vitreous adhesions are seen at the margins of the lesions. These lesions are also associated with round holes. If associated with traction , then tears are formed which cause detachment.

Over these lesions white interlacing lines are seen ophthalmologically. They are hyalinized blood vessels which forma cris cross pattern. These lesions enlarge circumferentially and new lesions also form. Most commonly involve the superior temporal quadrant bilaterally. Fluorescein angiography shows poor or absent perfusion in these areas.

LATTICE DEGENERATION



RETINITIS PIGMENTOSA IN MYOPIA



COMPLICATIONS:

1) Rhegmatogenous retinal detachment.
2) Choroid thrombosis and haemorrhages.
3) Cataract.
4) Severe visual impairment.
5) Chronic simple glaucoma.

CLINICAL EVALUATION FOR PATHOLOGICAL MYOPIA

1) **Visual acuity**

Is the most important criterion of testing the functional intergrity of the eye.

2) **Direct ophthalmoscopy**

Though the area observed is smaller, increased magnification obtained with this method allows detailed examination of the fundus.

3) **Indirect ophthalmoscope**

This technique is of special importance as it allows the examiner to form a clearer understanding of the cause. We can examine lesions involving fovea in the various pathologies and all features are documented in a retina chart.

4) **Fundus fluorescein angiography**

detects the posterior pole changes like SRNVM, Foster Fuch's spots, lacquer cracks and early macular hole in cases of pathological myopia.

5) **Indocyanine green angiography**

Is superior to FFA in studying choroidal lesions due to certain physical properties of ICGA dye. Choroidal circulation and areas of neovascularisation lying beneath the retina show much better with ICGA. Hyperfluroscence is seen with abnormal vessels or neovascularisation of the choroid and leakage of the disc. Also seen in areas of atrophy of pigment epithelium of the retina.

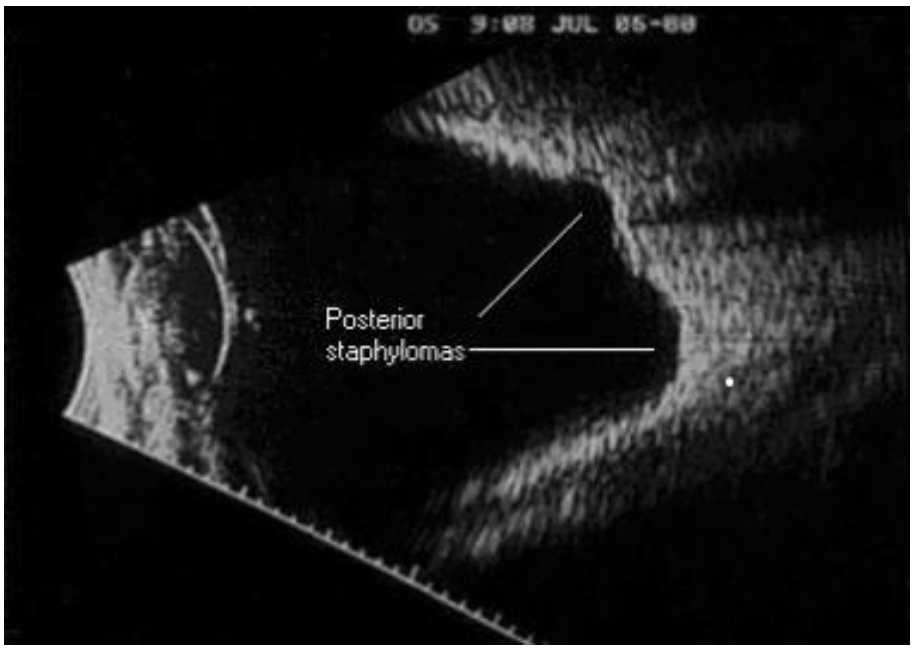
6) **A- scan**

It is a one dimensional scan display in which echoes are represented as vertical spikes from a baseline. This helps to differentiate axial myopia from lenticular myopia. A posterior staphyloma in high myopic eyes results in an increase in axial length.

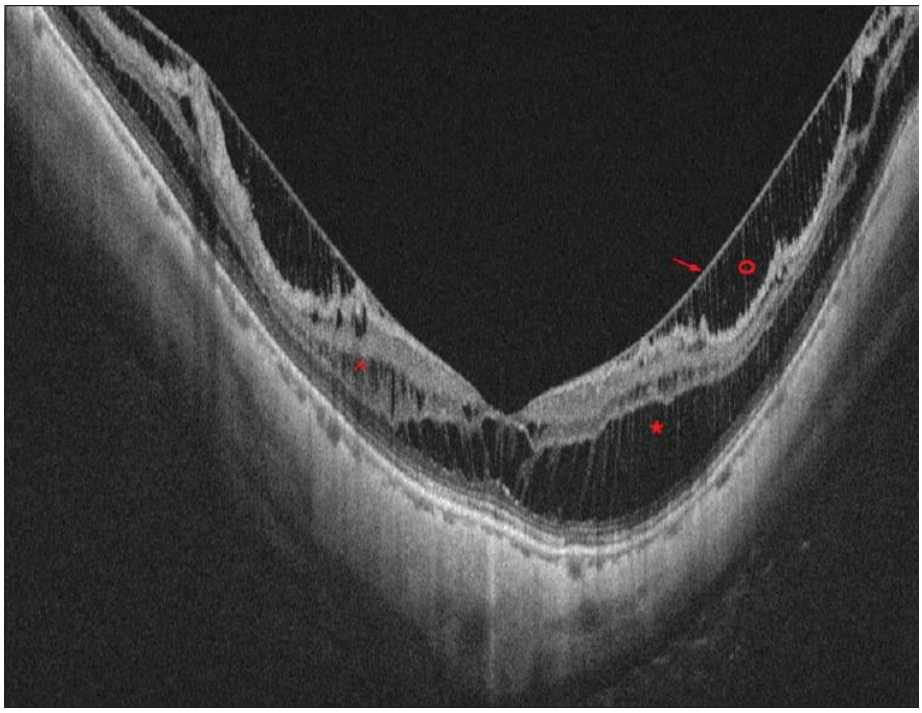
A-SCAN


PACSCAN plus
digital biometric ruler
AVG=23.57MM
SONOMED
Model 300A+
SONOMED

B-SCAN IN POSTERIOR STAPHYLOMA



OCT - RETINOSCHISIS



7) **Bscan**

Produces a two-dimensional acoustic section, hence echo is represented as a dot on the screen and not the spike. In high myopic eyes it is be used to evaluate the posterior segment and rule out the presence of retinal detachment and retinal tears. Posterior staphyloma is seen as a shallow excavation of the posterior pole with a smooth edge on sonographic evaluation of highly myopic eyes.

8) **Optical coherence tomography**

It is a new diagnostic technology which gives a cross sectional image of the retina in vivo. It has a high resolution similar to histological section by a light microscopy. OCT can be used to diagnose a foveal retinal detachment and a retinoschisis both of them are common features in severely myopic eyes with posterior staphyloma.

MANAGEMENT

Treatment of pathologic myopia may be divided into 3 goals – visual rehabilitation of the patient, prevention of myopic progression and the management of a variety of complicating diseases.

VISUAL REHABILITATION

I) Optical :

(i) Spectacles

Patients should be advised about the type of frame and the material of the lenses which are suitable for those patients with high myopia. High – index glass, plastic and polycarbonate lenses are suitable for high myopic patients. Special edge polishing and buffing can also improve lens cosmetics.

(ii) Contact lenses

Contact lenses are of special value in high myopia because they afford a dramatically improved appearance and enhance the visual acquity by reducing the image minification and expand the visual field. Both soft and gad-permeable contact lens designs are plausible. In cases of high myopia, it may be necessary to specify a minus-edge lenticular design to minimize the complications and discomfort of a thickened skirt.

II) SURGICAL :

Surgical correction of high myopia can be attempted through,

1) The flattening of corneal curvature for lower degrees.
2) Insertion of IOL into the phakic anterior chamber.
3) The removal of clear crystalline lens.
4) Shortening of axial diameter by scleral resection.
5) Role of LASIK in high myopia is controversial.

In cases of high myopia , the most useful low visin aid for distance is use of telescopic lens. New models with a small telescopic lens fitted into patients spectacles may be of great use.

III) OCULAR HYGIENE :

Ocular hygiene has undoubtedly greatly emphasized as an adjunct to control of myopic progression.

MANAGEMENT OF COMPLICATIONS

1) Retinal breaks and detachment

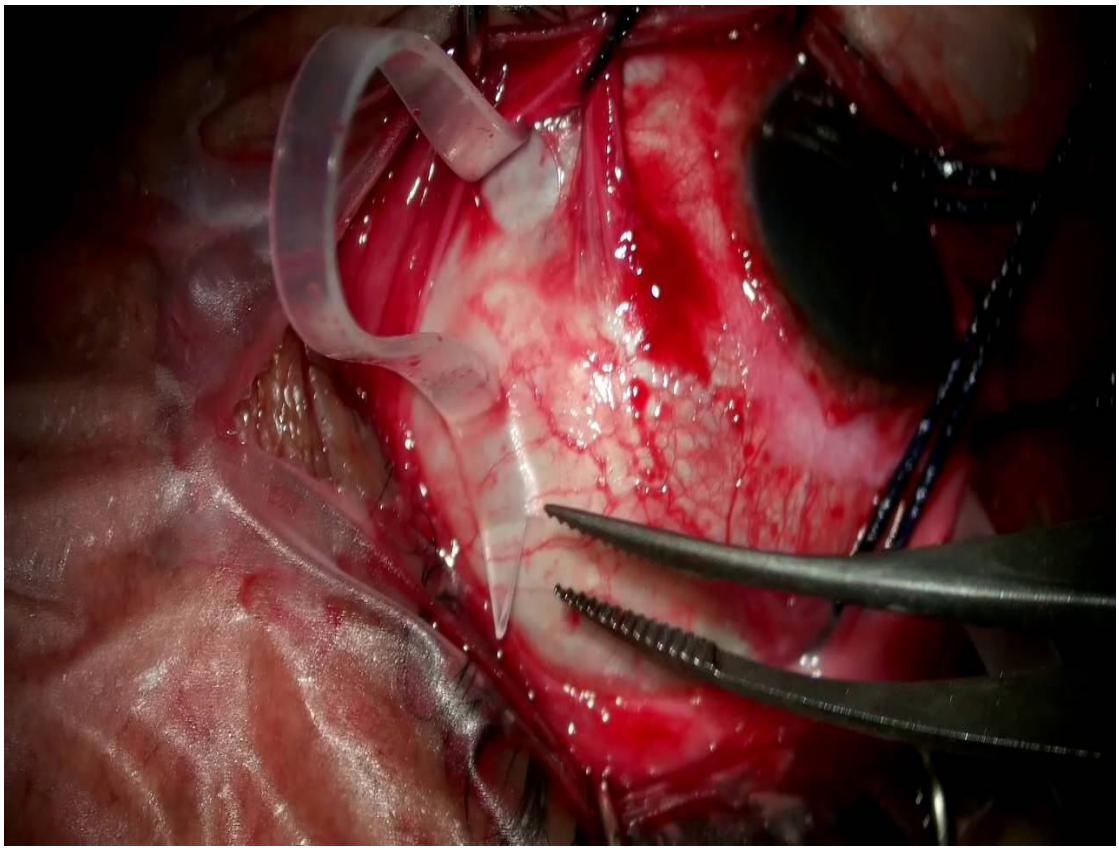
Treatment of retinal breaks is much rewarding thsn is, the attempted repair of an advanced detachmentYanoff has recommended the use of cryo retinal ablation prophylactically. Bensen & etal advised treating an adecuate margin of retina surrounding the lattice areas and then carrying out the treatment of ora. Retinal detachment surgery should

Be done taking into consideration of factors of scleral thinning and posterior staphyloma.

PRE-BARRAGE

POST - BARRAGE

RD SURGERY - EXTERNAL BUCKLING



2) Choroidal neovascular membrane

a) Extra foveal CNVM (> 200 micrometer from FAZ) - Green 514 nm / Red647 nm laser to cover CNVM

b) Juxta foveal CNVM (<200 micrometer &>1 micrometer from the center of FAZ) – Laser to cover CNV contigious blockage and 100 m beyond on non foveal side.

c) Subfoveal – Photodynamic theraphy.

3) Ocular hypertension and glaucoma management

The goal of glaucoma treatment is to preserve good visual function for thr patients life time. This can be attained by lowering the intraocular pressure to a level that will stop or atleast slow the progression of optic nerve damage and its consequent vision loss.

4) Management of cataract

Either phacoemulsification or SICS with proper IOL implantation has to be done taking proper precautions to prevent complications.

5) Management of strabismus and amblyopia.

Early squint correction is accepted as the most beneficial approach to congenital tropias associated with myopia. Appropriate spectacles and occlusion theraphy is advocated to manage amblyopia.

6) Management of retinitis pigmentosa

Low vision and genetic counselling.

7) Newer modality of treatment

Intravitreal injection of bevacizumab seems to be effective and safer treatment for macular CNVM.

PART II

AIM OF THE STUDY

1) **To evaluate the presence of fundus changes in patients with degenerative myopia.**
2) **To identify the predisposing factors.**
3) **The need for frequent follow ups.**
4) **Identification of high risk patients and appropriate management.**
5) **To rehabilitate refractory cases of high myopic patients with low visual aids and regular follow up**

MATERIALS AND METHODS

The study was carried out in Regional Institute of Ophthalmology in the Retina Clinic, Government Ophthalmic Hospital, Chennai.

Patient selection for the study was done by analyzing the medical record s of patients who came for follow up to our retina clinic with high myopia of at least - 6.00 diopters.

A detailed history of the patient, visual acuity assessment, intraocular pressure measurement, Slit lamp examination, colour vision and Fundus examination and fundus photography will be done.

Patients will be screened for the extent of retinal involvement. BCVA(Best corrected visual acuity), Intra-ocular pressure, Slit Lamp Examination and Fundus examination shall be done during follow up visits.

INCLUSION CRITERIA:

1) Patients with refractive error of more than

   6.00 diopters were selected for ophthalmologic examination .

2) Patients with an axial length of eyeball of more than 26 mm.

EXCLUSION CRITERIA:

1) Presence of cataract that justified visual impairment

2) History of previous ocular surgery.

3) Congenital retinal degenerations and dystrophies.

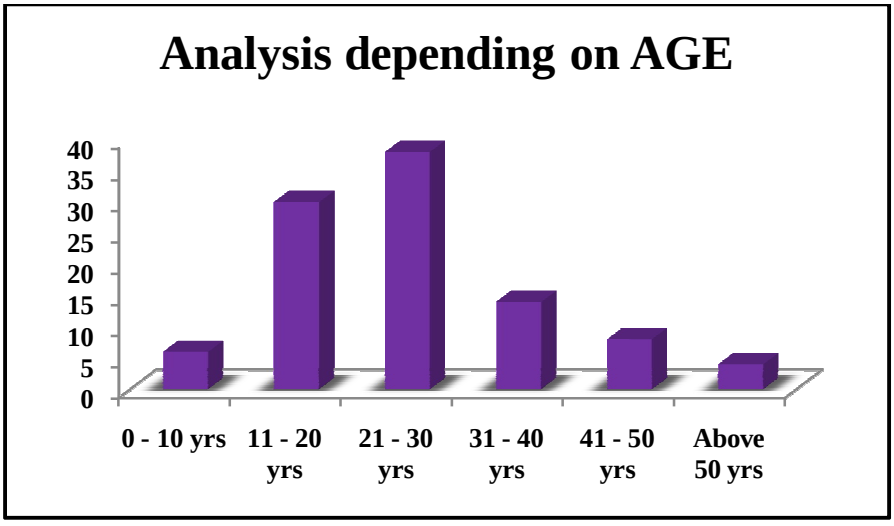
4) Patients with refractive errors less than – 6.00 diopters.

Observation & Analysis

OBSERVATIONS & ANALYSIS

1. ANALYSIS DEPENDING ON AGE

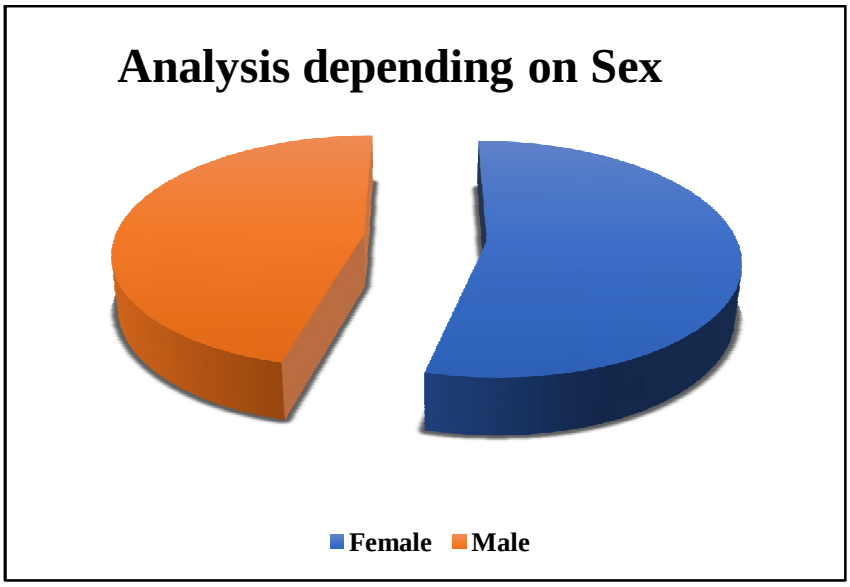
Age In	Frequency	Percentage
0 - 10 yrs	6	6%
11 - 20 yrs	30	30%
21 - 30 yrs	38	38%
31 - 40 yrs	14	14%
41 - 50 yrs	8	8%
Above 50 yrs	4	4%



Incidence of pathological myopia was found to be common in the age group of 21 to 30 years which correlates well with the **Framingham Eye Study Group**

2. ANALYSIS DEPENDING ON SEX

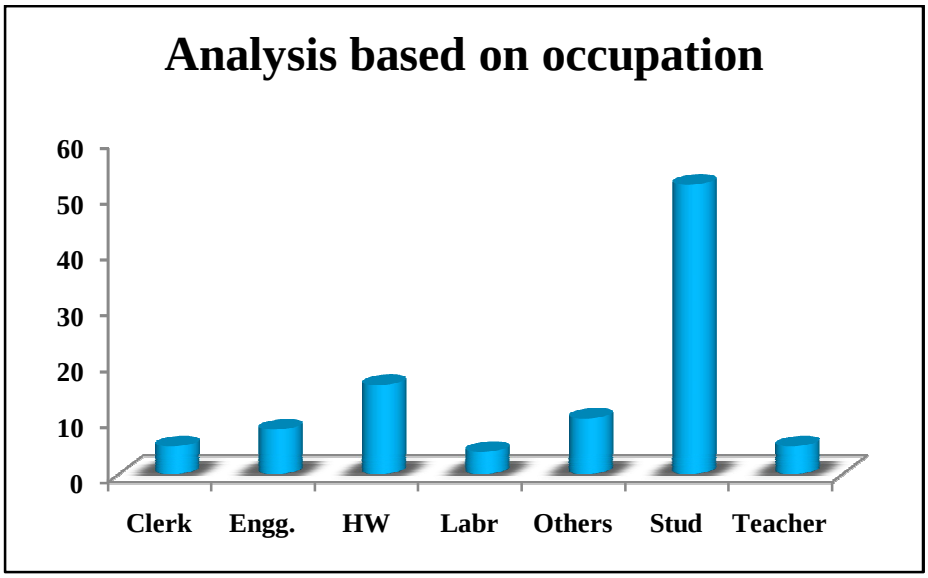
Gender	Frequency	Percent
Female	54	54%
Male	46	46%



Sex appears to have influence on incidence of myopia. Females have higher degree of degenerative changes in pathological myopia. **(Arun Verma et al)**

3. ANALYSIS BASED ON OCCUPATION

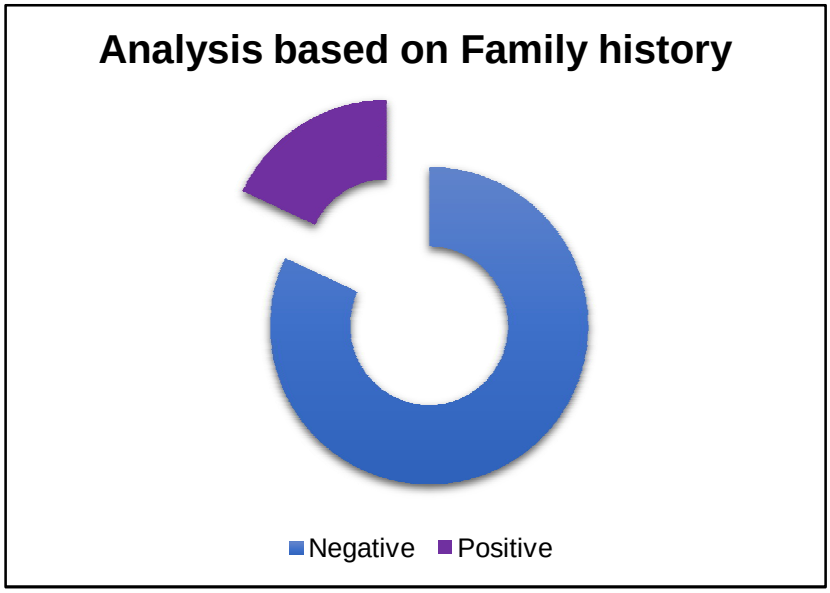
Occupation	No of Patients	Percentage
Clerk	5	5%
Engg.	8	8%
HW	16	16%
Labr	4	4%
Others	10	10%
Stud	52	52%
Teacher	5	5%



Majority patients in the study are from the student's community

4. ANALYSIS DEPENDING ON FAMILY HISTORY

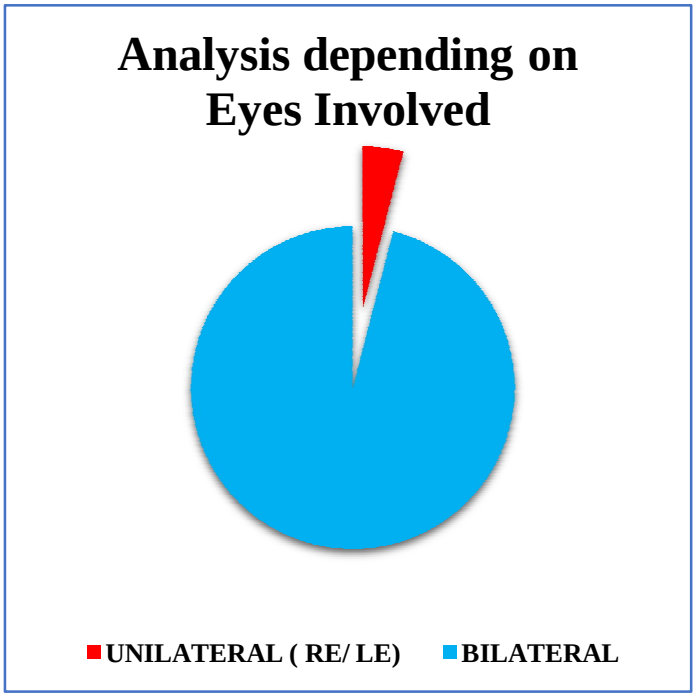
Total No of Cases	**100**
No of Cases with family history	18
Percentage	18%



Out of 100 cases examined only 18 cases were reported with the positive family history, which attributed to the lack of awareness in the low socio-economic group.

5. ANALYSIS DEPENDING ON EYES INVOLVED

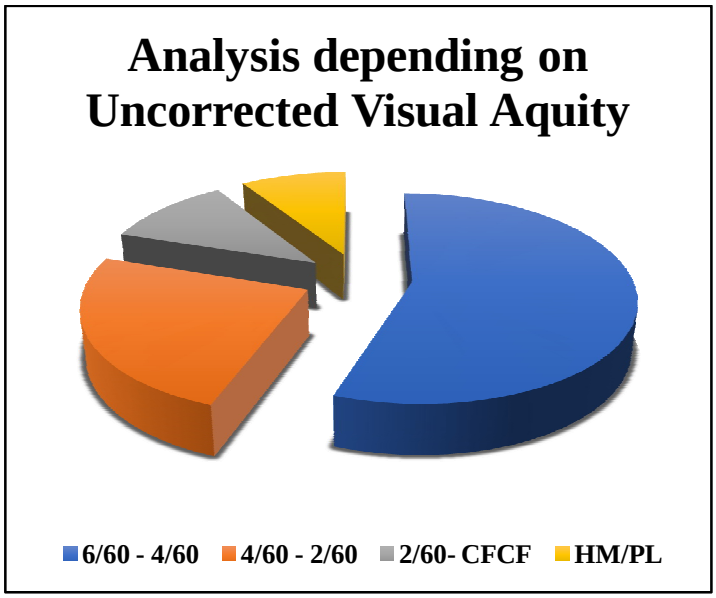
Eyes Involved	No of eyes	Percentage
UNILATERAL	8	4.0%
BILATERAL	192	96.0%



Out of 100 cases pathological myopia 96% has bilateral presentation and only 4% had unilateral presentation.

6. ANALYSIS DEPENDING ON UNCORRECTED VISUAL ACUITY

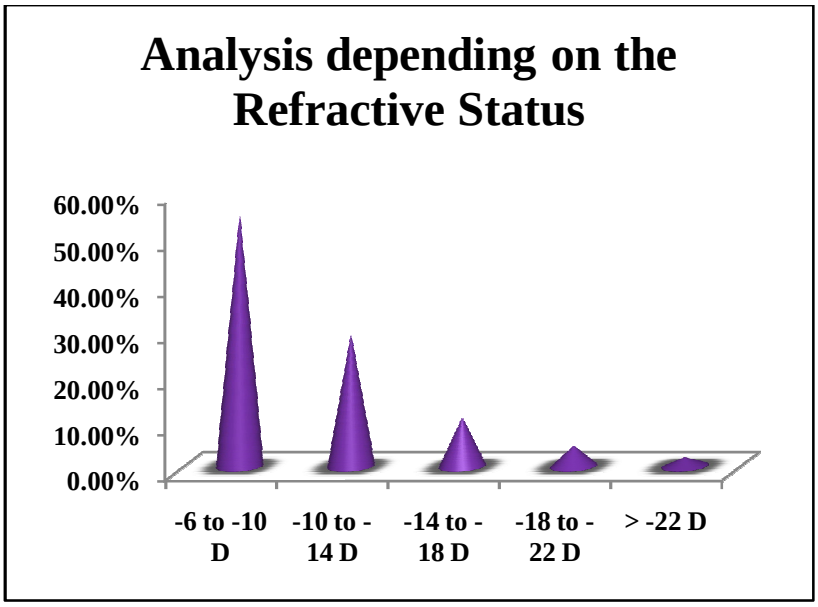
Visual Acuity	No of eyes	Percentage
6/60 - 4/60	111	55.5%
4/60 - 2/60	49	24.5%
2/60- CFCF	22	11%



Out of 200 eyes studied majority of the patients had a visual acuity that ranges from 6/60 to 4/60. That is closely followed by 4/60 to 2/60 group

7. ANALYSIS DEPENDING ON THE REFRACTIVE STATUS

Refractive Status	No of eyes	Percentage
-6 to -10 D	109	54.50%
-10 to -14 D	57	28.50%
-14 to -18 D	21	10.50%
-18 to -22 D	9	4.50%
> -22 D	4	2.00%

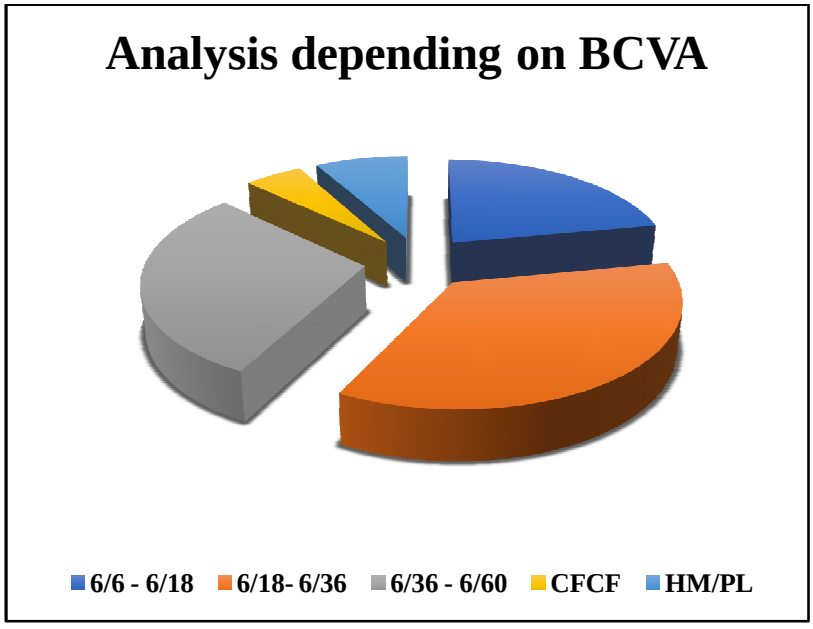


Among 100 patients analysed nearly 83% of the study group had a refractive index ranging from -6.0 D TO -14.0 D.

8. ANALYSIS DEPENDING ON THE BEST CORRECTED VISUAL ACQUITY.

BCVA	No of eyes	Percentage
6/6 - 6/18	44	22.0%
6/18- 6/36	71	35.5%
6/36 - 6/60	59	29.5%
CFCF	10	5.0%
HM/PL	16	8.0%

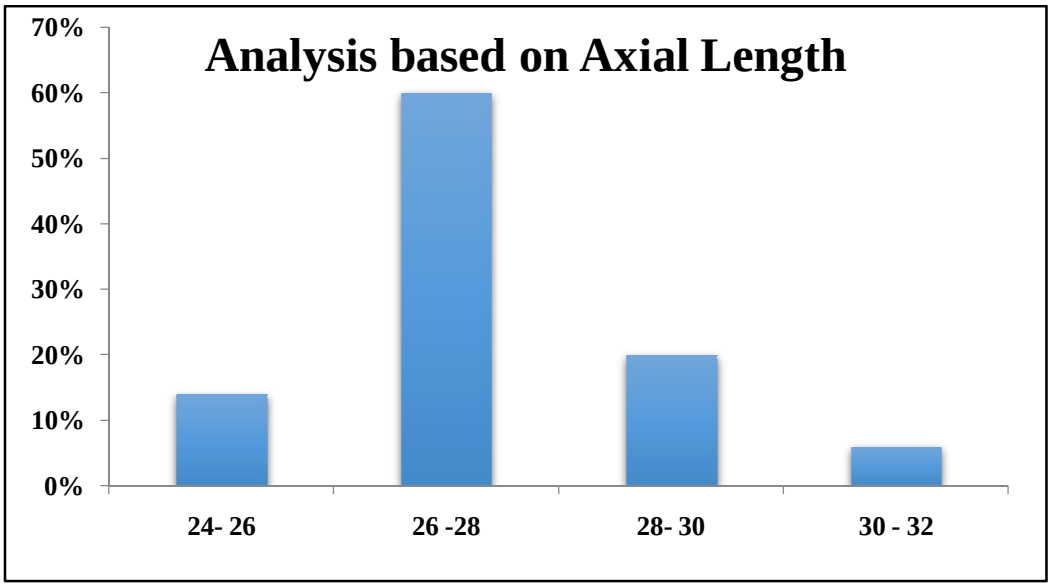
About 35.5% of patients had the best corrected visual acuity from 6/18 to 6/36, after doing proper retinoscopy and refraction. Visual correction in high myopic eye is obviously decreased when the dioptric value is higher.



9. ANALYSIS DEPENDING ON AXIAL LENGTH

10.

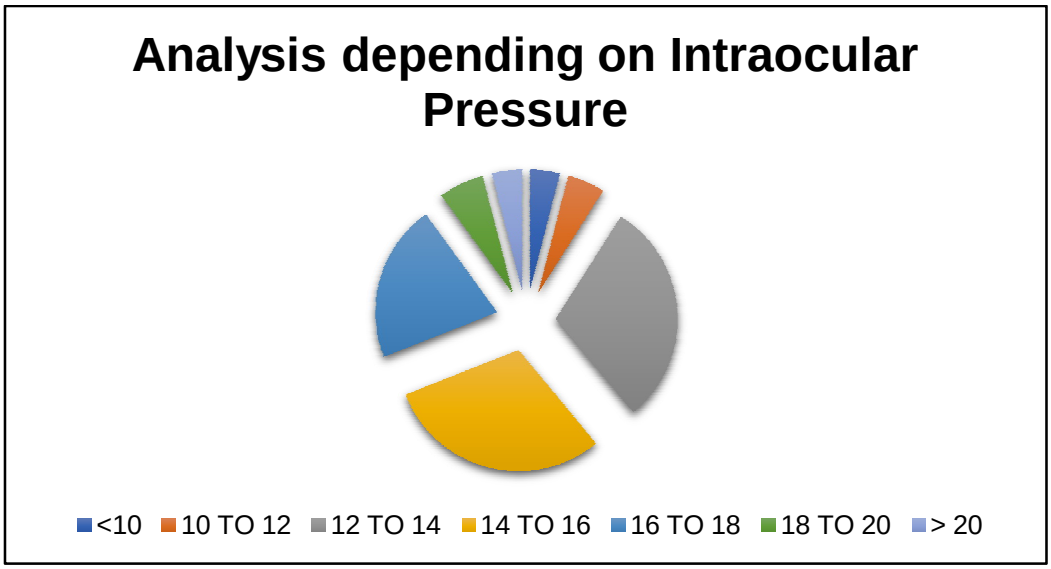
Axial Length (In mm)	No of eyes	Percentage
24- 26	28	14%
26 -28	120	60%
28- 30	40	20%
30 - 32	12	6%



Majority of the eyes included in the study had an axial length between the range of 26 mm to 28 mm. The progression of myopia depends mainly on the elongation of the eye ball.

10.ANALYSIS DEPENDING ON THE INTRAOCULAR PRESSURE.

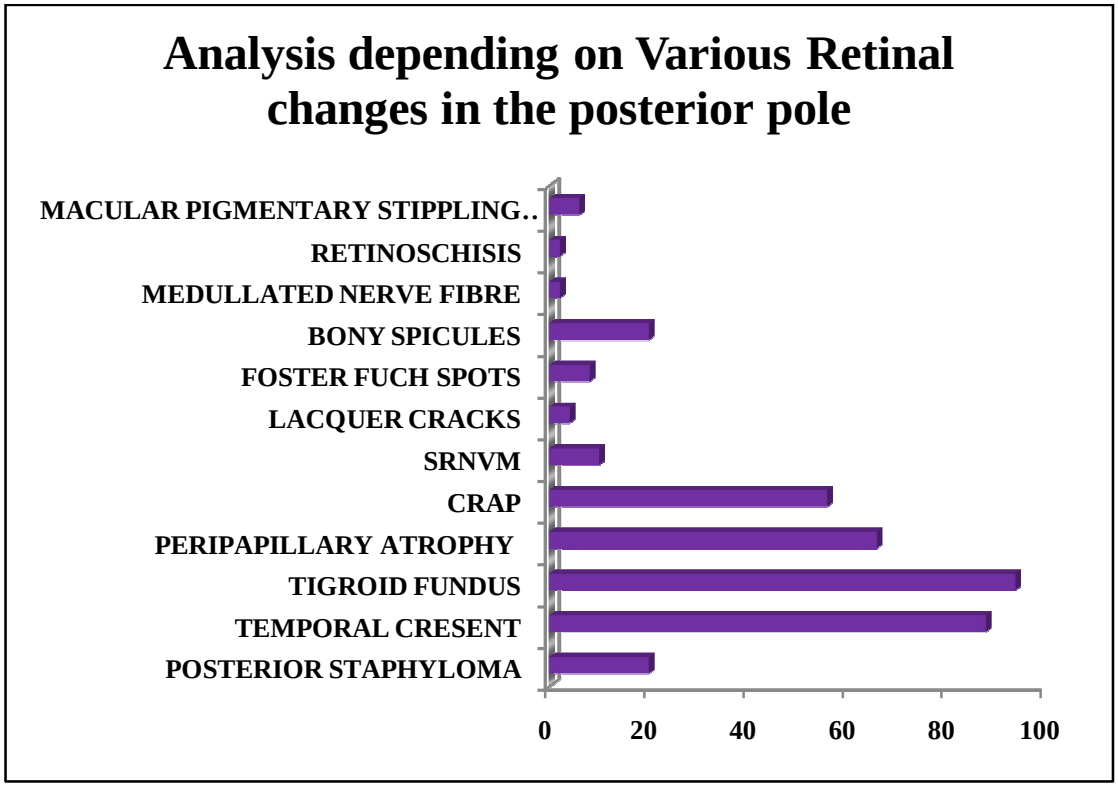
IOP	Number of Eyes	Percentage
<10	4	4%
10 TO 12	5	5%
12 TO 14	30	30%
14 TO 16	30	30%
16 TO 18	21	21%
18 TO 20	6	6%
> 20	4	4%



Out of the total 200 eyes taken in the study group, About 4% had an IOP of more than 20 mm of Hg by applanation tonometry. Nearly 92% had a normal IOP

11. ANALYSIS DEPENDING ON VARIOUS RETINAL CHANGES IN THE POSTERIOR POLE

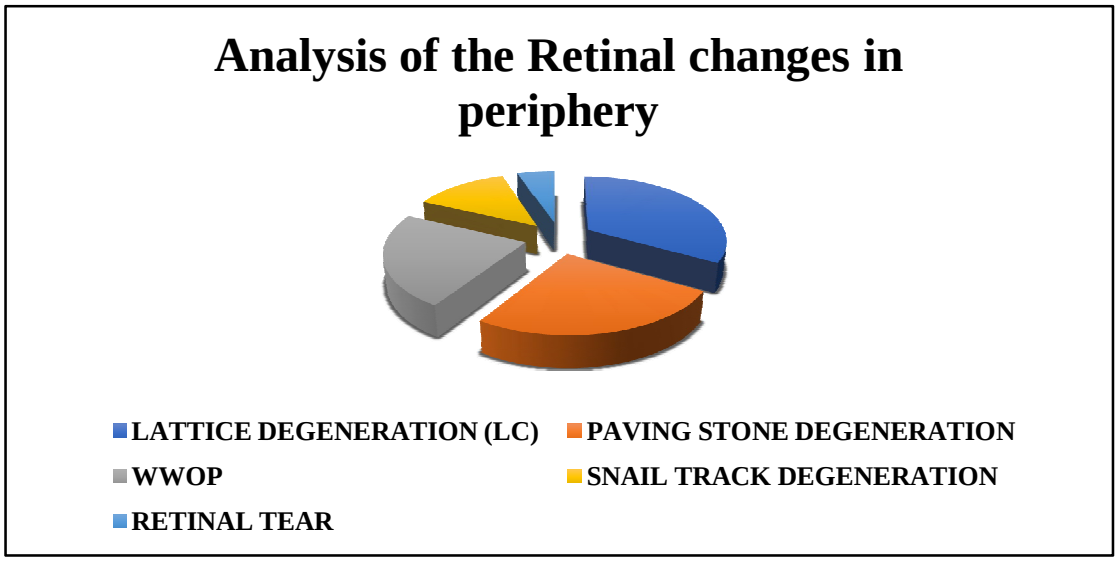
Retinal Changes	Frequency	Percentage
Posterior staphyloma	20	5.3%
Temporal cresent	88	23.4%
Tigroid fundus	94	25.0%
Peripapillary atrophy	66	17.5%
CRAP	56	14.9%
SRNVM	10	2.7%
Lacquer cracks	4	1.1%
Foster fuch spots	8	2.1%
Bony spicules	20	5.3%
Medullated nerve fibre	2	0.5%
Retinoschisis	2	0.5%
Macular pigmentary stippling	6	1.6%



Majority of the patients in the study group had temporal crescent and tigroid fundus as a common feature. 5.3% of patient had Posterior Staphyloma. Lacquer Cracks were seen in 1.1%. Foster Fuch Spots were seen in 2.1%. Chorioretinal atrophic patches were seen in 14.9% which corelated well with the study conducted by **Brasil et al.** SRNVM was seen in 2.7% of the patience which is in concordance of the study **Ohno – Matsui K**

12. ANALYSIS BASED ON THE RETINAL CHANGES IN THE PERIPHERY

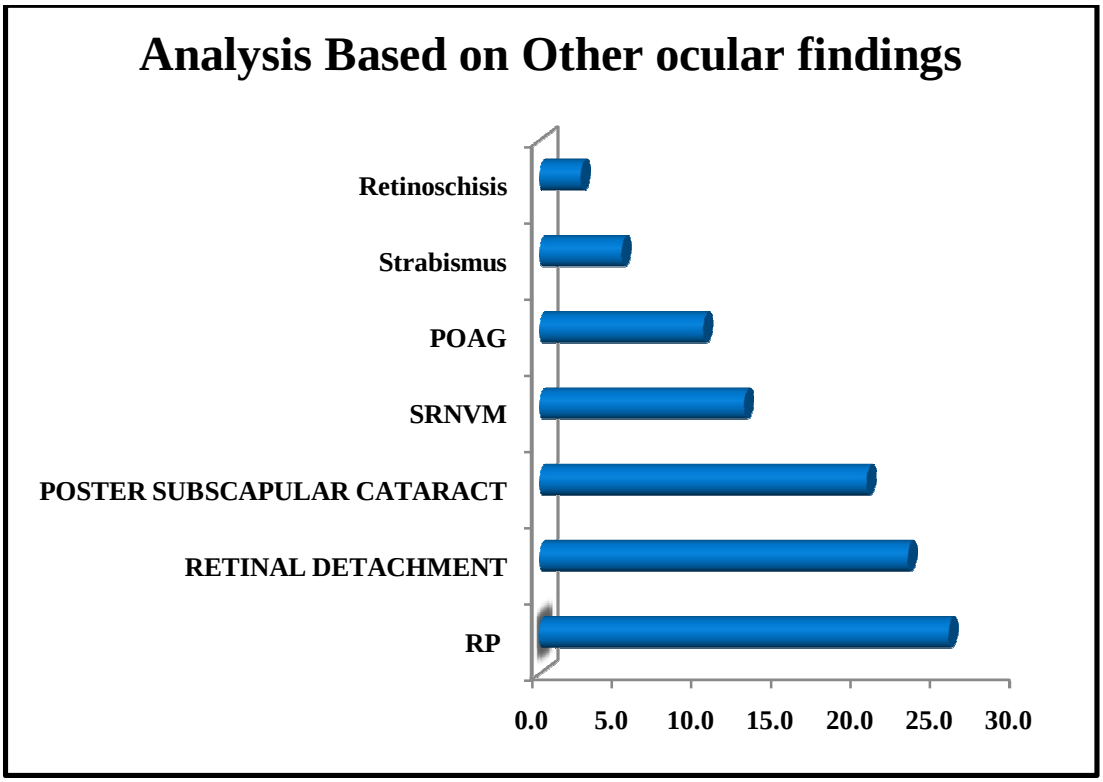
Retinal changes	Frequency	Percentage
Lattice Degeneration	26	33.3%
Paving Stone Degeneration	20	25.6%
WWOP	18	23.1%
Snail Track Degeneration	10	12.8%
Retinal Tear	4	5.1%



Lattice degeneration was found to be the commonest type of degeneration noted in the study, followed by Paving wave Degeneration which coincides well with the study by **Celorio, Preutt R C**.

13. ANALYSIS OF OTHER OCULAR FINDINGS

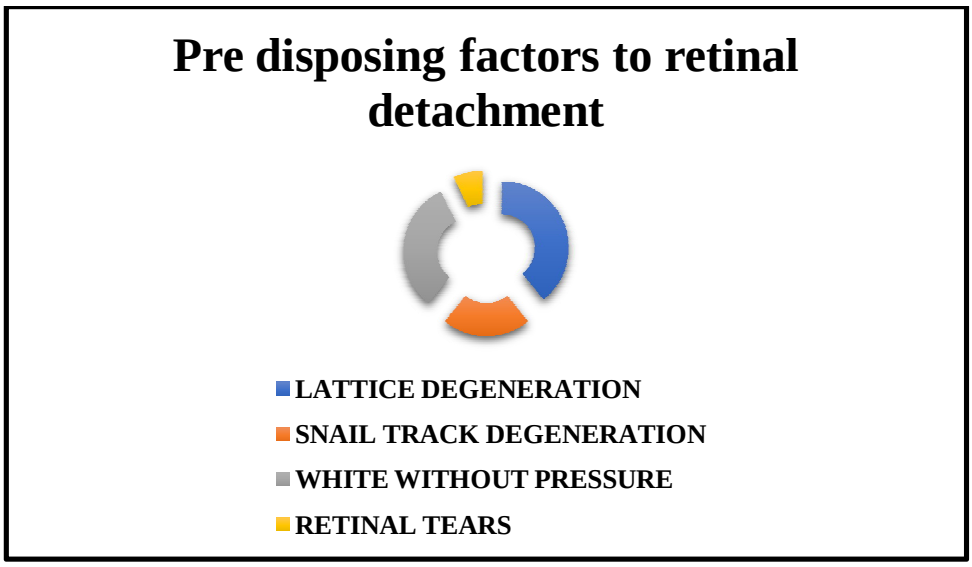
Conditions	No of Eyes	Percentage
RP	20	25.6%
Retinal detachment	18	23.1%
Postr subscapular cataract	16	20.5%
SRNVM	10	12.8%
POAG	8	10.3%
Strabismus	4	5.1%
Retinoschisis	2	2.6%



Higher incidents of pigmentary dystrophy (25.6%) is noted in the study group which is followed by Retinal Detachment (23.1%). Other associations noted were posterior sub capsular cataract. **(Beaver Dam Eye Study),** increased intra ocular pressure **(Blue Mountain Study)** and Strabismus (5.1%)

14. CONDITIONS PREDISPOSING TO RETINAL DETACHMENT

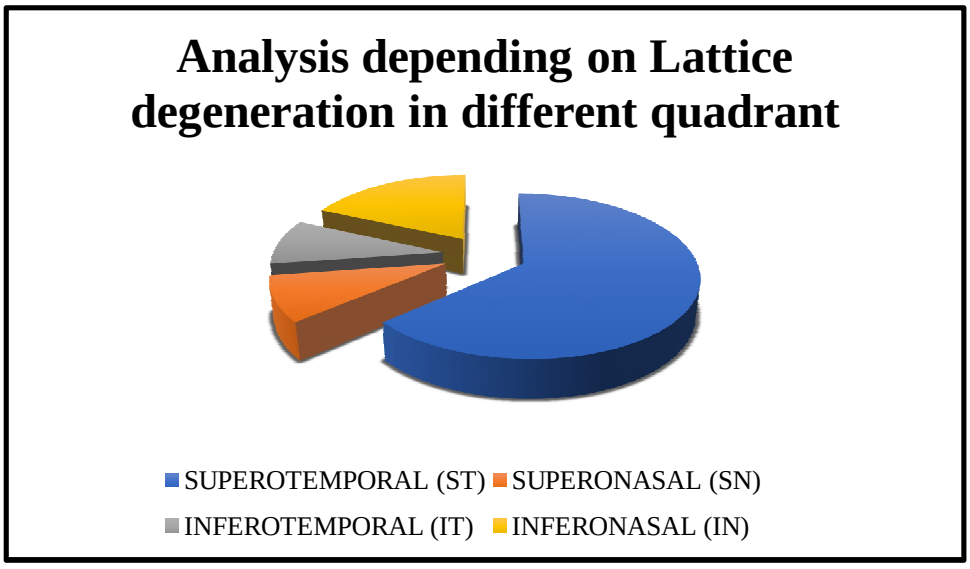
Peripheral Degenerations	Number of Eyes	Percentage
LATTICE DEGENERATION	22	39.29%
SNAIL TRACK DEGENERATION	12	21.43%
WHITE WITHOUT PRESSURE	18	32.14%
RETINAL TEARS	4	7.14%



Lattice degeneration with hole was found to be the commonest degeneration among the pre disposing factors for retinal detachment, which is followed by White without Pressure.

15. ANALYSIS DEPENDING ON THE LATTICE DEGENERATION IN DIFFERENT QUADRANTS

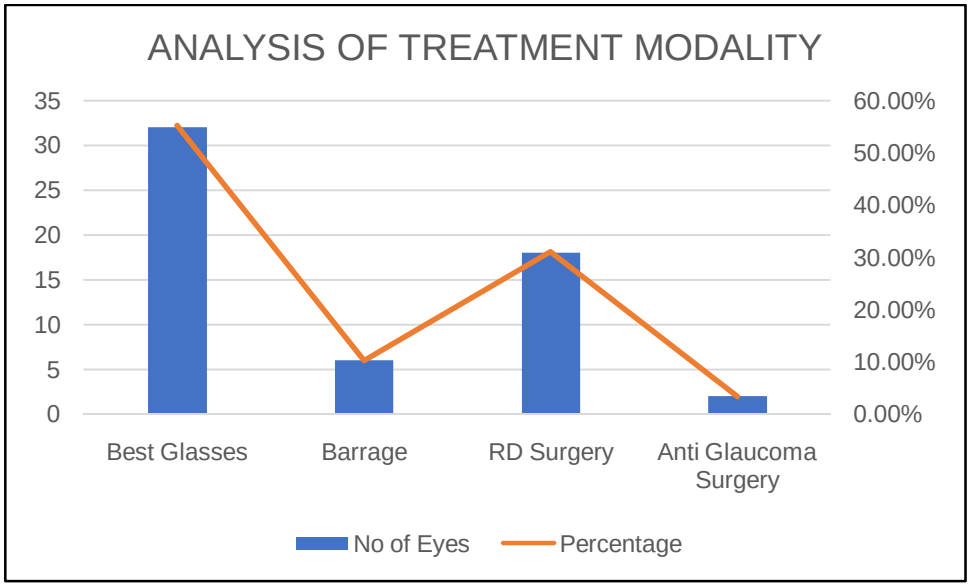
Quadrant	Number of Eyes	Percentage
SUPEROTEMPORAL (ST)	14	63.64%
SUPERONASAL (SN)	2	9.91%
INFEROTEMPORAL (IT)	2	9.91%
INFERONASAL (IN)	4	18.18%



Lattice degeneration was most commonly located in the supero temporal Quadrant of the Retina (63.64%)

16. ANALYSIS BASED ON THE TREATMENT MODALITY

Treatment Modality	Number of Eyes	Percentage
Best Glasses	32	55.17%
Barrage	6	10.34%
RD Surgery	18	31.03%
Anti-Glaucoma Surgery	2	3.45%



Based on the analysis, prescription of glasses for myopia was found to be the most common treatment modality followed by RD Surgery and Barrage Laser. Anti-Glaucoma surgery are done for specific cases.

DISCUSSION

100 patients with pathological myopia were studied, of which the incidence was common between the age group 21 to 30 years, which correlated well with the **Framingham eye study** that suggested that aging along with the mechanical stretching is also important for the development of the fundus changes.

- Sex appears to have influence on the incidence of myopia. Females are more prone for the degeneration in myopia and myopia per say.
- Only 18% of cases had family history of myopia, majority did not have any positive family history. The decreased incidence may be due to lack of the awareness in low socio economic group.
- Majority of the study group where from student community. This suggests that the people are aware of the medical condition early and seek medical help early.
- Out of 100 cases of pathological myopia 96% has bilateral presentation where as only 4% had unilateral presentation. Timely and consistent therapy in unilateral myopia for uniocular and binocular visual acuity has better prognosis.
- About 70 percentage of myopia fall within the diopteric range of -6 to -14D, which stats that extreme degrees of myopia are <more frequent.

- Majority of the eyes studied in the study has axial length of 27-28 mm, that indicates that the axial elongation of the eye ball is the main component causing myopic progression**.(Liull et al)**
- In the study about 4% had an increase in the IOP of more than 20 mmhg by applanation tonometry**.(Blue mountain study)**
- Out of 200 eyes even with the full correction with glasses, majority of them 59 eyes BCVA improved only to 6/36-6-60, which suggest that higher the diopteric power of the eye, harder the vision can be ideally corrected. More the pathologic change in the posterior pole, greater is the severity of the damage**.(Journal of eye science: 2003 Dec 19(4) 211-4)**
- Patients in the study group mostly had temporal cresent and tessellated fundus as a common feature followed by posterior staphyloma 5.7% , SRNVM -2.7% Foster fuchs spots 2.1% and lacquercracks 1.1%, which correlates well with Brasil et al **(Araq.Bras Ophthal Mar-April, 69 (2) 203-6)**
- Lattice degeneration is the commonest peripheral degeneration noted in the study. The amount of axial elongation influences the prevalence of the lattice.
- Myopic patients has a higher risk of the development of glaucoma compared to a non myopic patient.
- The associations with posterior subcapsular cataract, strabismus , retinal detachment and retinitis pigmentosa are also noted as per
- The most common predisposing factor leading to the development of RD was found to be lattice with hole followed by paving stone degeneration.

- The most common location of the lattice was noted in the superotemporal quadrant due to the probable cause of excessive stretching and increased vascularity in this area.
- The risk of RD showed to be increased in the patients with higher degree of myopia of more than -10 D.
- Therefore, this study demonstrates that the fundus findings of a high myopic patient was more in the posterior pole. This information may be useful in evaluating the following patients with a moderate to high degree of myopia post-surgical refractive modification.

SUMMARY

- Out of 200 with pathological myopia were analysed based on the ocular fundus changes in the posterior pole , axial length and the corneal curvature.
- Highest incidence of pathological myopia was noted to be in the age group of 21-30 Years.
- The majority of the patients had no significant family history. 4% had the unilateral myopia on their presentation
- The higher prevalence of myopia in the student population shows that the most common environmental factor like the increasing education and higher amount of near work.
- About 55% patients had an UCVA ranging between 6/60 – 4/60
- 70 % of the patients with pathological myopia where in the range of -6 to -14 dioptres. Very high degree of myopia were less frequent.
- There is a definite correlation between the increase in the axial length and the high degree of myopia.
- Higher the refractive power, more difficult is to achieve the normal vision. This establishes the fact that pathological changes in posterior pole of eye is responsible for the defective vision.
- Lattice degeneration is the most common degeneration whish is predominantly found in the supero temporal quadrant.
- Temporal cresent and the tessellated fundus was found as a common feature in majority of the patients.

- Posterior staphyloma was found in 5.3% eyes.
- Lacquer crackes was found in 1.1% and foster fuch in 2.1% of eyes.
- Choroidal neovascularisation was seen in 12.8%eyes.
- Retinitis pigmentosa, retinal detachment , glaucoma , posterior subcapsular cataract were the most common associations.
- Hence the study demonstrates the various factors that may be useful while evaluating and following up of patients with moderate and high degrees of myopia.

CONCLUSION

Pathological myopia is a complex disease of the eye in which the patient presents not only the visual morbidity but also have a diseased eye. Therefore they have to be approached according to their needs and presentations.

All cases of myopia must be examined meticulously with the indirect ophthalmoscope as they are usually associated with degenerative changes. Indirect ophthalmoscope pick up the complications at the earliest and can be treated effectively thereby aiding in retaining of the useful ocular function. The awareness should be created among the myopic individuals regarding the safety precautions, visual hygiene, risks and complications involved.

Patient should be well informed regarding the warning signs and symptoms for early and better management. Patients with pathological myopia must be monitored periodically.

Genetic counselling and low vision aids are advised whenever necessary.

PART III

BIBLIOGRAPHY

TEXTBOOKS

1. Apple , DS (Mosby) ocular pathology – Myopia – Pathology (37-42)
2. Albert & Jakobiec – Principles & practice o ophthalmology 3 edition.
3. Curtin B.J- The Myopias, Basic science & clinical management : Philadelphia (Harper & row)
4. David Abrams : Duke elder's practice of refraction (Elsevier science , India)
5. Guyer David .R, Yanuzzi Lawrence A; Retina, Vitreous – Macula, WB saunders Sectionn- 2, Macular disorders
6. HARJ,Jr,WH – Adlers physiology of the eye : clinical application (Harcourt brace & company , Asia)
7. Kanski jackJ : clinical ophthalmology B/H 8 th edition
8. Peyman, GA Saunders , DR Goldberg : Principles & practice of ophthalmology Vol 2
9. Stephen – J- Ryan – Retina (vol 2) medical retina , vol 3 surgical retina.
10. Snell RS, Lemp Michae A – Clinical Anatomy of the eye (Blackwell Science)
11. Ramajit Sihota, Radhika Tandon – Parsons disease of the eye.
12. Myron Yanoff, Jay's Duker – Ophthalmology (mosby)
13. Sir stewart Duke -Elder-System of ophthalmology, ophthalmic optics & refraction.

14. Curtin BJ. The myopias: Basic science and clinical management. Philadelphia, PA: Harper and Row; 1985.

15. Lin LL, Chen CJ, Hung PT, Ko LS. Nation-wide survey of myopia among schoolchildren in Taiwan, 1986. Acta Ophthalmol Suppl. 1988;185:29–33

16. Wang Q, Klein B, Klein R, Moss S. Refractive status in the Beaver Dam Eye Study. Invest Ophthalmol Vis Sci. 1994;35:4344–4347

17. Duke□Elder S. Pathological refractive errors. System of ophthalmology. Vol V. Ophthalmic optics and refraction. St Louis: Mosby, 1970297–373.

18. Avila M P, Jalkh A E, Mainster M A. *et al* Natural history of choroidal neovascularization in degenerative myopia. Ophthalmology 1984911573–1581

19. Mitchell P, Hourihan F, Sandbach J. *et al* The relationship between glaucoma and myopia: the Blue Mountains Eye Study. Ophthalmology 19991062010–2015

20. Virgili G, Menchini F, Virgili G. Laser photocoagulation for choroidal neovascularisation in pathologic myopia. Cochrane Database Syst Rev 200519CD004765

21. National Society for the Prevention of Blindness Estimated statistics on blindness and vision problems. New York: National Society for the Prevention of Blindness, 196644

22. Rabb M F, Garoon I, LaFranco F P. Myopic macular degeneration [review]. Int Ophthalmol Clin19812151–69.

23. Ikuno Y, Gomi F, Tano Y. Potent retinal anterior traction as a possible cause of myopic foveoschisis. Am J Ophthalmol 2005139462–467

24. Levy J H, Pollock H M, Curtin B J. The Fuchs' spot: an ophthalmoscopic and fluorescein angiographic study. Ann Ophthalmol 197791433–1443.

25. Noble K G, Carr R E. Pathologic myopia. Ophthalmology 1982891099–1100.

26. Grossniklaus H E, Green W R. Pathologic findings in pathologic myopia. Retina 199212127–133.

27. Takashima T, Yokoyama T, Futagami S, Ohno-Matsui K, Tanaka H, Tokoro T, et al. The quality of life in patients with pathologic myopia. Jpn J Ophthalmol. 2001;45:84–92

28. Zadnik K, Satariano WA, Mutti DO, Sholtz RI, Adams AJ. The effect of parental history of myopia on children's eye size. JAMA. 1994;271:1323–1322.

29. Dunphy EB, Stoll MR, King SH. Myopia among American male graduate students. Am J Ophthalmol. 1968;65:518–521.

PROFORMA

1.NAME :

2.AGE/SEX :

3.OP NO :

4.ADDRESS:

5.OCCUPATION:

6.CHIEF COMPLAINTS:

A.OCULAR

RE LE

Duration

a.Defective Vision (Distant/Near)

b.Total loss of vision

c. Floaters

d.Flashes

e.Positive scotoma

f.Macropsia/Micropsia/Metamorphopsia

g.Field defects

h.Pain

i.Redness

B.SYSTEMIC- Joint pains/ rashes/fever/ oral ulcers/genital ulcers/ skin lesions/ chest pain cough/ weight loss/ weakness of limbs/others

7.PAST HISTORY:

Diabetes Mellitus/ Hypertension/ Ischemic heart Disease/ Tuberculosis/ Bronchial Asthma/ HIV/Syphilis/ skin diseases like psoriasis

Duration:

8.FAMILY HISTORY:

9.PERSONAL HISTORY:

Smoking/ Alcoholism/ Vegetarian/ Non Vegetarian/ education/ hours of study time/reading distance/

10.TREATMENT HISTORY :

11.EXAMINATION: **RE** **LE**

Visual Acuity

Extra Ocular Examination

Tension

Lids

Conjunctiva

Cornea

Iris

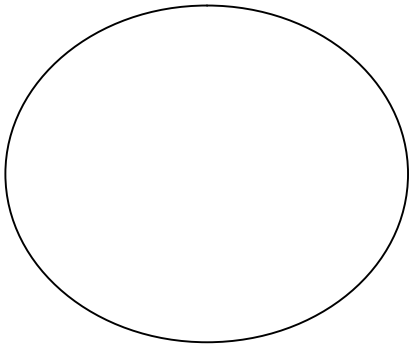
Anterior Chamber

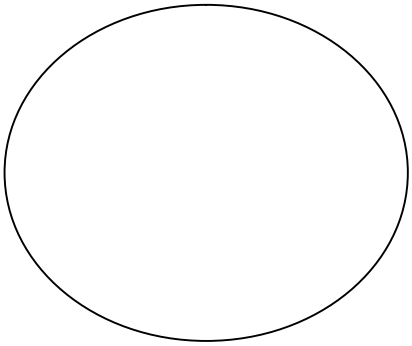
Pupil

Lens

Vitreous

12. FUNDUS EXAMINATION:





RE **LE**

13. CLINICAL DIAGNOSIS :

14. FUNDUS PHOTOGRAPHY :

15. A- SCAN

16. K- READING

17.CORNEAL DIAMETER.

18.ASSOCIATED FINDINGS :

1) Keratoconus.

2) Glaucoma.

3) Retinitis Pigmentosa

15.TREATMENT.

Medical:

1) Glasses.

2) Low vision aids.

Surgery:

1) Barrage laser.

2) Retinal Detachment surgery.

3) Vitrectomy.

4) Phakic IOL.

5) LASIK.

16. FOLLOW UP:

KEY TO MASTER CHART

F/H	**–**	**Family History**
VA	**–**	**Visual Acuity**
Ref. Power	**–**	**Refractive Power**
BC/VA	**–**	**Best Corrected / Visual acuity**
AL	**–**	**Axial Length.**
F. Changes	**–**	**Fundus Changes**
RE	**–**	**Right Eye**
LE	**–**	**Left Eye**
Wnl	**–**	**Within Normal Limits**
VF	**–**	**Vitreous Floaters**
Tess	**–**	**Tesselation**
Temp	**–**	**Temporal**
LD	**–**	**Lattice Degeneration**
STD	**–**	**Snail Tract Degeneration.**
PSD	**–**	**Paving Stone Degeneration**
PVD	**–**	**Posterior Vitreous Detachment**

PS	**–**	**Posterior Staphyloma**
RT	**–**	**Retinal Tear**
RD	**–**	**Retinal Detachment**
WWP	**–**	**White With Pressure**
WWOP	**–**	**White Without Pressure**
STQ	**–**	**Supero Temporal Quadrant**
SNQ	**–**	**Superonasal Quadrant.**
INQ	**–**	**Infero Nasal Quadrant**
ITQ	**–**	**Infero temporal quadrant**
SQ	**–**	**Superior quadrant**
BG	**–**	**Best glasses**
EXP	**–**	**Explant**
Pro.Cryo	**–**	**Prophylactic cryotheraphy**
PR	**–**	**periodic review**
PPC	**–**	**Posterior polar cataract.**
CRAP	**–**	**Chorioretinalatropic patch**

Sl.No	Name	Age	Sex	Occup	F/H	UCVA		Tension in mm of hg		Laterality		Refractive power in diopters		BCVA		AX.LENGTH		Kreading		Post Retinal Changes		Periretinal Changes		Associati on	Treatme nt
						RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE		
1	Suresh	21	M	Stud	+	CFCF	4/60	Tlow	18	+	+	-8.00/-3.00 90°	-10.00	6/60	6/18	26.0	26.9	-47.25	+47.5	RD+RD SX Done	Large MP,TC	RD	TS,LD+ST OF with ATH		RD Sx LE-barrage
2	Sasikala	50	F	HW	-	CFCF	CFCF	13	15	+	+	-23.00	-23.00	6/60	CFCF	31.5	31.6	+44.00	+44.50	PS,TF,TC, PPA	TF,TC,PP A				BG,PR
3	Manikan dan	36	M	Clerk	-	4/60	HM+	15	13	+	+	-9.00/-2.00 90°	—	6/18	HM+	27.9	27.4	+46.[illegible]	+46.25	PS,PPA,C RAP	SRNVM			LE-SRNVM	Referred
4	Malar	30	F	HW	-	5/60	6/60	24	25	+	+	-10.00	-9.00	6/24	6/36	27.8	27.5	+45.50	+45.25	CDRatio 0.6 TC,TF,NV	CDRatio 0.6 TC,TF,NV			BE-POAG	MED RX
5	Devikrish nan	35	F	Cerg	+	5/60	4/60	16	16	+	+	-7.00	-7.5	6/36	6/60	26.4	26.8	+44.25	+44.25	View Hazy d/t PSC	View Hazy d/t PSC	Retinoshi sis		BE-PSC	SICS with PR
6	Vijayaraj	26	M	Stud	-	PL+	1/60	Tlow	18	+	+	—	-22.00	PL+	6/60	28.9	30.8	+46.00	+46.25	No view	PPA,CRA P,PS	No view	WWOP	B Scan RD RE	RE-RD Sx
7	Lakshmi	11	F	Stud	-	3/60	5/60	13	13	+	+	-9.00	6.00/-0.5 90°	6/18	6/12	26.5	25.9	+45.50	+45.75	PS,TC	TF,TC	PVS	PVS		
8	Ranjitha	9	F	Stud	-	6/6	6/60	15	16	-	-	+/-	7.00	6/6	6/12	23.9	25.1	+47.25	+45.25	N	TF,MC	N, RE			
9	Nisha	8	F	Stud	-	5/60	6/5	14	13	+	-	-8.00	+/-	6/18	4/6	26.8	23.4	+47.50	+47.00	Medullat ed N Fibres	N		N		BG, PR
10	Priya	24	F	HW	+	5/60	4/60	17	16	+	+	-7.50/-2.00 90°	-8.00/-2.50 90°	6/36	6/36	25.6	26.8	+44.75	+44.75	PD,PF,PP A,BS	PD,PF,PP A,BS			RP	BG, PR
11	Abirecial	16	F	Stud	-	4/60	5/60	14	14	+	+	-9.50	-8.00	6/36	6/36	26.9	26.5	+45.00	+45.00	TF,TC,MP S	TF,TC				BG,PR
12	Bala	12	F	Stud	-	2/60	2/60	15	16	+	+	-11.00	-11.00	6/60	6/60	27.8	27.7	+44.75	+44.75	FFS,PPA	TF,TC,PP A				BG,PR
13	Barani	19	M	Stud	-	5/60	5/60	18	18	+	+	-8.00	-8.5	6/9	6/24	26.5	26.9	+45.75	+45.75	N	TF,TC		STD		BG,PR
14	Badri	24	M	Engg.	-	2/60	HM+	13	Tlow	+	+	-10.50	—	6/36	HM+	27.1	27.5	+44.50	+44.00	TF,TC	RD	LD (STQ)	RD		LE-RD Sx,RE-Pro,cryo
15	Deepa	15	F	Stud	-	6/60	6/60	14	14	+	+	-6.00	-6.50	6/12	6/12	26.5	26.8	+44.75	+44.75	TF,TC,BS, PD,AA	TF,TC,BS, PD,AA			RP	BG,PR
16	Divya	22	F	HW	-	2/60	3/60	17	17	+	+	-8.50/-2.50 90°	-8.00/-2.00 90°	6/36	6/36	27.0	26.7	+46.25	+46.25	TC,MPS	PPA,TCL C,CRAP,T F		PVS		
17	Vijay	28	M	Clerk	-	5/60	CFCF	14	Tlow	+	+	-7.00	—	6/36	CFCF	26.3	26.5	+46.50	+46.25	PPA,CRA P	RD	PVS	RD		RD Sx - EXP
18	Sathish	12	M	Stud	-	1/60	2/60	18	17	+	+	-10.50	-10.00	6/60 NIP	6/60	27.5	26.9	+46.25	+46.25	BS,PD,AA ,TC,TF	BS,PD,AA ,TC,TF			RP	
19	Kapil	28	M	Stud	+	5/60	5/60	14	14	+	+	-8.00/-3.50 90°	-8.50/-3.00 90°	6/36 NIP	6/36	25.8	25.9	+47.25	+47.25	TC,TF	CRAP,TF	WWOP	WWOP		BG, PR
20	Manjula	27	F	HW	-	3/60	6/6	17	18	+	-	-10.00	+/-	6/36	6/6	27.3	23.9	+46.50	+46.75	FFA, MPS, PPA	WNL				PR, RE-CL
21	Naveen	45	M	Engg.	-	3/60	HM	16	16	+	+	-8.50	—	6/60	HM+	26.8	26.5	+46.00	+46.50	MC,TF,CR AP	SRNVM,T F,CRAP				
22	Rajesekа r	16	M	Stud	-	4/60	5/60	13	13	+	+	-10.50	-10.00	6/36	6/60	27.2	27.5	+44.00	+44.50	PPA,CRA P	PPA,CRA P	LD (STQ), Hole	STD		RE-Barrage
23	Anitha	9	F	Stud	-	2/60	2/60	15	16	+	+	-11.50	-12.00	6/18	6/60	27.5	28.0	+46.50	+46.50	WNL	PPA,MD				BG,PR
24	Ashokku mar	13	M	Stud	+	1/60	CFCF	15	16	+	+	-13.50	-14.00	6/36	6/60	28.5	28.7	+44.25	+44.50	CRAP,PP A	TS,TF,TC, PPA				BG,PR
25	Preethi	24	F	Stud	-	CFCF	6/60	10	14	+	+	—	-6.50/-1.00 180°	CFCF	6/9	26.2	26.3	+45.50	+46.00	—	TF,TC	RD,LD			RE-RD Sx with Cryo
26	Suresh	20	M	Stud	-	3/60	4/60	16	18	+	+	-11.50	-11.00	6/36	6/24	28.1	27.8	+46.50	+46.50	FFS,PPA, TF,CRAP, TC	LC,PPA,T F				BG,PR
27	Mani	17	M	Stud	-	CFCF	CFCF	14	14	+	+	-22.00	-23.50	6/60	5/60	31.5	32.1	+44.50	+44.50	PPA,PS,T F,TC	PPA,PS,T F,TC				BG,PR
28	Valli	35	F	HW	-	6/6	5/60	19	17	-	-	+/-	-10.50	6/6	6/36	24.2	27.1	+44.50	+44.50	WNL	TF,TC				BG,PR

29	Vasanthi	32	F	Lect	+	CFCF	2/60	16	15	+	+	—	-14.50	CFCF	6/36	27.8	28.5	+45.00	+45.50	Retinoschisis	PPA,TC,TF	LD-IT,PVS	LD-IT,PVS		
30	Indhumathi	12	F	Stud	-	3/60	4/60	13	14	+	+	-13.00	-12.50	6/18	6/36	28.1	27.8	+45.50	+45.00	CRAP,TF	CRAP,TF		LD-SN		Prophylactic Cryo - LE
31	Nandhini	14	F	Stud	-	1/60	2/60	19	16	+	+	-12.00	-12.50	6/36	6/24	27.4	27.9	+44.25	+44.50	PS,PPA	PS,PPA	PVS,STD	PVS,STD	Strabismus	Sx-done BG
32	Swarnam	51	F	HW	-	CFCF	1/60	16	15	+	+	-15.00	-14.50	6/60	5/60	29.1	28.8	+45.25	+45.25	View Hazy, TF	View Hazy, TF,CRAP			PSC-BE	Sics with IOL
33	Ravikumar	13	M	Stud	-	3/60	4/60	14	13	+	+	-11.50	-11.00	6/24	6/36	27.6	27.4	+45.00	+44.75	CRAP,TC	CRAP,TC	LD,STQ-Hole	LD,STQ-Hole		LE-ARC, RE-Barrage
34	Pratheep	33	M	Driver	-	2/60	3/60	20	20	+	+	-8.50	-8.00	2/60	6/60	26.9	26.5	+45.00	+45.00	CRAP,SRNVM, TC, TF	MD, TC, TF, CRAP			SRNVM - RE	Referred
35	Thendral	27	F	Stud	+	HM+	4/60	12	13	+	+	—	-9.00	HM+	6/36	27.2	27.0	+45.25	+45.50		CRAP,TC	RD	WWOP		RD-Sx
36	Anbuselvi	19	F	Stud	-	3/60	3/60	15	18	+	+	-12.00	-11.00	6/60	6/60	27.2	27.0	+46.50	+46.25	PS,CRAP, TF	CRAP,TF	STD		RP	
37	Mythili	12	F	Stud	-	4/60	3/60	17	18	+	+	-10.50	-8.00	6/24	6/18	28.1	27.9	+44.50	+44.00	BS,PD,AA	BS,PD,AA				RE-Barrage, LE- RD Sx
38	Prabhakaran	26	M	Prof	-	4/60	3/60	19	20	+	+	-11.00	-12.00	6/12	6/18	27.1	26.8	+45.25	+45.25	TC,PPA	TC,PPA	LD,STQ-Hole	Shallow INF RD		
39	Vennilla	23	F	Stud	-	3/60	2/60	18	16	+	+	-18.50	-19.50	6/24	6/36	27.4	27.8	+44.00	+44.25	PPA,TC, TF	PPA,TC, TF	WWOP		Strabismus	
40	Balaji	38	M	Labr	-	5/60	5/60	26	28	+	+	-10.00	-13.50	6/24	6/60 NIP	29.5	30.1	+45.50	+45.25	CD Ratio 0.5, TF, MC	CD Ratio 0.5, TF, MC		WWOP	BE-POAG	AG Sx
41	Vikram	25	M	Stud	-	2/60	HM+	16	10	+	+	-15.00	—	6/60	HM+	27.1	27.3	+46.00	+46.25	PPA	Bullous RD	PVS	RT with RD		RD Sx & Cryo
42	Jeeva	21	F	Stud	-	1/60	2/60	18	14	+	+	-19.00	-17.50	5/60	6/60	27.5	28.1	+44.75	+44.50	BS,PD,AA	BS,PD,AA	STD	LD INQ	RP	BG,PR
43	Murugan	28	M	Engg.	-	3/60	3/60	17	16	+	+	-11.00	-9.50	6/36	6/24	29.0	28.7	+46.25	+46.25	CRAP	CRAP, FFS	WWOP			BG,PR
44	Sekar	58	M	WM	+	1/60	3/60	14	15	+	+	-16.00	-14.00	1/60 NIP	3/60	26.8	28.7	+47.25	+47.50	View Hazy - PSC	View Hazy - PSC		WWOP	BE-PSC	SICS-IOL IMP
45	Praveena	35	F	TR	-	1/60	6/60	16	14	+	+	—	-6.50	1/60 NIP	6/18	25.8	26.0	+46.25	+46.50	SRNVM, CRAP	TC,TF	PVS	WWOP	RE-SRNVM	Referred
46	Sandeep	36	M	TR	-	3/60	2/60	15	16	+	+			6/60	6/20	28.1	29.0	+46.50	+46.50	View Hazy, CRAP, PPA	View Hazy, CRAP, PPA			BE-PSE	SICS - IOL
47	Sivakami	21	F	Labr	-	3/60	HM+	14	11	+	+	-11.00	—	6/36	HM+	28.2	27.9	+47.50	+47.00	CRAP,TF, PPA	CRAP,TF, PPA	LD STQ with Hole	RD		RD-Sx
48	Subramanian	24	M	Engg.	-	3/60	PL+	13	10	+	+	-12.50	—	6/60	PL	28.5	27.5	+46.25	+46.25	PPA, TC, TF	PPA, TC, TF	WWOP	RT with RD		RD Sx
49	Venkatesan	42	M	TR	-	3/60	CFCF	16	17	+	+	-14.50	-16.00	6/12	6/12	29.1	28.4	+47.25	+47.50	CRAP, TC, TF	TC, TF				BG, PR
50	Radhika	49	F	HW	+	HM+	2/60	17	18	+	+	—	-20.00	CFCF	6/60	24.1	25.6	+44.50	+44.50	SRNVM, PPA, TC	TC, TF		LD-STQ	RE-SRNVM	LE-PR, RE-Laser
51	Ravi	21	M	Stud	+	CFCF	4/60	Tlow	18	+	+	-8.00/-3.00 90°	-11.00	6/36	6/18	26.0	25.8	+47.25	+47.5	RD+RD SX Done	Large MP,TC	RD	TS,LD+STQT with ATH		RD Sx LE-barrage
52	sasireka	50	F	HW	-	CFCF	CFCF	13	15	+	+	-23.00	-23.00	6/60	CFCF	31.5	31.6	+44.00	+44.50	PS,TF,TC, PPA	TF,TC,PPA				BG,PR
53	ragurama n	36	M	Clerk	-	4/60	HM+	15	13	+	+	-9.00/-2.00 90°	—	6/18	HM+	27.9	27.4	+46.25	+46.25	PS,PPA,CRAP	SRNVM			LE-SRNVM	Referred
54	kavitha	30	F	HW	-	5/60	6/60	24	25	+	+	-10.00	-9.00	6/24	6/36	27.8	27.5	+45.50	+45.25	CDRatio 0.5 TC,TF,NV	CDRatio 0.7 TC,TF,NV			BE-POAG	MED RX

55	dhanam	35	F	Cang	+	5/60	4/60	16	16	+	+	-8.00	-7.5	6/36	6/60	26.4	26.8	+44.25	+44.25	View Hazy d/t PSC	View Hazy d/t PSC			BE-PSC	SICS with PR
56	guna	26	M	Stud	-	PL+	1/60	Tlow	18	+	+	—	-18.00	PL+	6/60	28.9	30.8	+46.00	+46.25	No view	PPA,CRA P,PS	No view	WWOP	B Scan RD RE	RE-RD Sx
57	dharini	11	F	Stud	-	1/60	5/60	13	13	+	+	-9.00	6.00/-0.5 90°	6/18	6/12	26.5	25.9	+45.50	+45.75	PS,TC	TF,TC	PVS	PVS		
58	suji	9	F	Stud	-	6/6	6/36	15	16	-	+	+/-	-7.00	6/6	6/12	23.9	25.1	+47.25	+45.25	N	TF,MC	N, RE			
59	nethra	7	F	Stud	-	5/60	6/6	14	13	+	-	-6.00	+/-	6/18	6/6	26.8	23.4	+47.50	+47.00	Medullated N Fibres	N		N		BG, PR
60	ragini	24	F	HW	+	4/60	4/60	17	17	+	+	-7.50/-2.00 90°	-8.00/-2.00 A1.Z10 290°	6/36	6/24	25.6	26.8	+44.75	+44.75	PD,PF,PPA,BS	PD,PF,PPA,BS			RP	BG, PR
61	Meera	16	F	Stud	-	4/60	5/60	14	14	+	+	-8.50	-8.00	6/36	6/36	26.9	26.5	+45.00	+45.00	TF,TC,MPS	TF,TC				BG,PR
62	sheela	32	F	Stud	-	2/60	3/60	15	16	+	+	-11.00	-11.00	6/60	9/60	27.8	27.7	+44.75	+44.75	TF5,PPA	TF.TC.PPA				BG,PR
63	murugan	19	M	Stud	-	5/60	5/60	18	18	+	+	-8.00	-8.5	6/9	6/24	26.5	26.9	+45.75	+45.75	N	TF,TC		STD		BG,PR
64	kesavan	24	M	Engg.	-	2/60	HM+	13	Tlow	+	+	-10.50	—	6/36	HM+	27.1	27.5	+46.50	+44.00	TF,TC	RD	LD (STQ)	RD		LE-RD Sx,RE-Pro, cryo
65	rani	15	F	Stud	-	6/60	6/60	14	14	+	+	-6.60	-6.50	6/12	6/12	26.5	26.8	+44.75	+44.75	TF,TC,BS, PD,AA	TF,TC,BS, PD,AA			RP	BG,PR
66	aradhana	22	F	HW	-	2/60	1/60	17	17	+	+	-8.50/-2.50 90°	-8.00/-2.00 90°	6/36	6/36	27.0	26.7	+46.25	+46.25	TC,MPS	PPA.TC.LC,CRAP,TF		PVS		
67	marimuthu	28	M	Clerk	-	5/60	CFCF	14	Tlow	+	+	-7.00	—	6/36	CFCF	26.3	26.5	+46.50	+46.25	PPA,CRAP	RD	PVS	RD		RD Sx - EXP
68	krishnamoorthy	12	M	Stud	-	1/60	2/60	18	17	+	+	-10.50	-10.00	6/60 NIP	6/60	27.5	26.9	+46.25	+46.25	BS,PD,AA,TC,TF	BS,PD,AA,TC,TF			RP	
69	sampath	28	M	Stud	+	5/60	5/60	14	14	+	+	-8.00/-3.50 90°	-8.50/-3.00 90°	6/36 NIP	6/36	25.8	25.9	+47.25	+47.25	TC,TF	CRAP,TF	WWOP	WWOP		BG, PR
70	Manju	27	F	HW	-	3/60	6/6	17	18	+	-	-10.00	+/-	6/36	6/6	27.3	23.9	+46.50	+44.75	FFA, MPS, PPA	WNL				PR, RE-CL
71	prabhu	45	M	Engg.	-	3/60	HM	16	16	+	+	-8.50	—	6/60	HM+	26.8	26.5	+46.00	+46.50	MC,TF,CRAP	SRNVM,TF,CRAP				
72	Rajakumar	16	M	Stud	-	4/60	5/60	13	13	+	+	-10.50	-10.00	6/36	6/60	27.2	27.5	+44.00	+44.50	PPA,CRAP	PPA,CRAP	LD (STQ), Hole	STD		RE-Barrage

73	pratheba	9	F	Stud	-	2/60	2/60	15	16	+	+	-11.50	-12.00	6/18	6/60	27.5	28.0	+46.50	+46.50	WNL	PPA,MD				BG,PR
74	kumaran	13	M	Stud	+	1/60	CFCF	15	16	+	+	-13.50	-14.00	6/36	6/60	28.5	28.7	+44.25	+44.50	CRAP,PPA	TS,TF,TC, PPA				BG,PR
75	savitha	24	F	Stud	-	CFCF	6/60	10	14	+	+	—	-6.50/-1.00 180°	CFCF	6/9	26.2	26.3	+45.50	+46.00	—	TF,TC	RD,LD			RE-RD Sx with Cryo
76	ram	20	M	Stud	-	3/60	4/60	16	18	+	+	-11.50	-11.00	6/36	6/24	28.1	27.8	+46.50	+46.50	FFS,PPA, TF,CRAP, TC	LC,PPA,TF				BG,PR
77	sethu	17	M	Stud	-	CFCF	CFCF	14	14	+	+	-22.00	-23.50	6/60	5/60	31.5	32.1	+44.50	+44.50	PPA,PS,TF,TC	PPA,PS,TF,TC				BG,PR
78	febi	25	F	HW	-	6/6	5/60	19	17	-	+	+/-	-10.50	6/6	6/36	24.2	27.1	+44.50	+44.50	WNL	TF,TC				BG,PR
79	vanathi	32	F	Lect	+	CFCF	2/60	16	15	-	+	—	-14.50	CFCF	6/36	27.8	28.5	+45.00	+45.50	Retinoschisis	PPA,TC,TF	LD-IT,PVS	LD-IT,PVS		
80	Madhimozhi	12	F	Stud	-	3/60	4/60	13	14	+	+	-13.00	-12.50	6/18	6/36	28.1	27.8	+45.50	+45.00	CRAP,TF	CRAP,TF		LD-SN		Prophylactic Cryo - LE
81	meri	14	F	Stud	-	3/60	2/60	19	16	-	+	-12.00	-12.50	6/36	6/24	27.4	27.9	+44.25	+44.50	PS,PPA	PS,PPA	PVS,STD	PVS,STD	Strabismus	Sx-done BG
82	rekha	51	F	HW	-	CFCF	1/60	16	15	+	+	-15.00	-14.50	6/60	5/60	29.1	28.8	+45.25	+45.25	View Hazy, TF	View Hazy, TF,CRAP			PSC-BE	Sics with IOL
83	kishore	13	M	Stud	-	3/60	4/60	14	13	+	+	-11.50	-11.00	6/24	6/36	27.6	27.4	+45.00	+44.75	CRAP,TC	CRAP,TC	LD,STQ-Hole	LD,STQ-Hole		LE-ARC, RE-Barrage
84	arul	33	M	Driver	-	2/60	3/60	20	20	+	+	-8.50	-8.00	2/60	6/60	26.9	26.5	+45.00	+45.00	CRAP,SRNVM, TC, TF	MD, TC, TF, CRAP			SRNVM - RE	Referred
85	Hema	27	F	Stud	+	HM+	4/60	12	13	+	+	—	-9.00	HM+	6/36	27.2	27.0	+45.25	+45.50		CRAP,TC	RD	WWOP		RD-Sx
86	Selvi	19	F	Stud	-	3/60	3/60	15	18	+	+	-12.00	-11.00	6/60	6/60	27.2	27.0	+46.50	+46.25	PS,CRAP, TF	CRAP,TF	STD		RP	
87	Megala	12	F	Stud	-	4/60	3/60	17	18	+	+	-10.50	-8.00	6/24	6/18	28.1	27.9	+44.50	+44.00	BS,PD,AA	BS,PD,AA				RE-Barrage, LE- RD Sx
88	Chandru	26	M	Prof	-	4/60	3/60	19	20	+	+	-11.00	-12.00	6/12	6/18	27.1	26.8	+45.25	+45.25	TC,PPA	TC,PPA	LD,STQ-Hole	Shallow INF RD		
89	Nila	23	F	Stud	-	3/60	2/60	18	19	+	+	-18.50	-19.50	6/24	6/36	27.4	27.8	+44.00	+44.25	PPA,TC, TF	PPA,TC, TF	WWOP		Strabismus	
90	Balaji	38	M	Labr	-	5/60	5/60	26	28	+	+	-10.00	-13.50	6/24	6/60 NIP	29.5	30.1	+45.50	+45.25	CD Ratio 0.5, TF, MC	CD Ratio 0.5, TF, MC		WWOP	BE-POAG	AG Sx
91	Raja	25	M	Stud	-	2/60	HM+	16	18	+	+	-15.00	—	6/60	HM+	27.1	27.3	+46.00	+46.25	PPA	Bullous RD	PVS	RT with RD		RD Sx & Cryo
92	Jeevan	21	F	Stud	-	1/60	2/60	18	14	+	+	-19.00	-17.50	5/60	6/60	27.5	28.1	+44.75	+44.50	BS,PD,AA	BS,PD,AA	STD	LD INQ	RP	BG,PR
93	Moorthy	28	M	Engg.	-	3/60	3/60	17	16	+	+	-11.00	-9.50	6/36	6/24	29.0	28.7	+46.25	+46.25	CRAP	CRAP, FFS	WWOP			BG,PR
94	Balaji	58	M	WM	+	1/60	3/60	14	15	+	+	-16.00	-14.00	1/60 NIP	3/60	26.8	28.7	+47.25	+47.50	View Hazy - PSC	View Hazy - PSC		WWOP	BE-PSC	SICS-IOL IMP
95	Raji	35	F	TR	-	1/60	6/60	16	14	+	+	—	-6.50	1/60 NIP	6/18	25.8	26.0	+46.25	+46.50	SRNVM, CRAP	TC,TF	PVS	WWOP	RE-SRNVM	Referred
96	krishakaran	36	M	TR	-	3/60	2/60	15	16	+	+			6/60	6/20	28.1	29.0	+46.50	+46.50	View Hazy, CRAP, PPA	View Hazy, CRAP, PPA			BE-PSE	SICS - IOL
97	Sivakalai	21	F	Labr	-	3/60	HM+	14	11	+	+	-11.00	—	6/36	HM+	28.2	27.9	+47.50	+47.00	CRAP,TF, PPA	CRAP,TF, PPA	LD STQ with Hole	RD		RD-Sx
98	Santosh	24	M	Engg.	-	3/60	PL+	13	10	+	+	-12.50	—	6/60	PL	28.5	27.5	+46.25	+46.25	PPA, TC, TF	PPA, TC, TF	WWOP	RT with RD		RD Sx
99	Bharathi	42	M	TR	-	3/60	CFCF	16	17	+	+	-14.50	-16.00	6/12	6/12	29.1	28.4	+47.25	+47.50	CRAP, TC, TF	TC, TF				BG, PR
100	Radhu	49	F	HW	+	HM+	2/60	17	18	+	+	—	-20.00	CFCF	6/60	24.1	25.6	+44.50	+44.50	SRNVM, PPA, TC	TC, TF		LD-STQ	RE-SRNVM	LE-PR, RE-Laser